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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—16TH YEAR. SYDNEY, SATURDAY, JANUARY 26, 1929. No. 4.

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FOOD DEFICIENCY DISEASES: THEIR CAUSE AND PREVENTION.[1]

By J. S. PURDY, D.S.O., V.D., M.D., C.M. (Aberdeen), D.P.H. (Cambridge), F.R.S.E., F.R.G.S.,
Metropolitan Officer of Health, Sydney.

We lay the blame of our illness now upon the air, now upon the unhealthfulness of the place, or the length of the journey, to take it off from the intemperance and luxury which was the cause of it.

Plutarch.

Observe
The rule of "not too much" by temperance taught
In what thou eat'st and drink'st, seeking from thence
Due nourishment, not gluttonous delight.

Milton.

DEFICIENCY diseases are conditions characterized by symptoms due to the absence of some vital accessory food factor or vitamin essential to growth or virility. These diseases are quite distinct from conditions merely the result of malnutrition.

Previous to the present century the study of nutrition was concerned mainly with the chemical analysis of foods, digestion, assimilation and energy metabolism. It is only recently that a biological investigation of foods based on their physiological functions has been made by actual feeding experiments. The calorific value in proteins, carbohydrates, fats and salts is now supplemented by investigation of their nutritional value. By studying experimentally the effect of any definite diet on the life history of animals and the experience of man in regions where geographically or from other circumstances the diet is restricted, monotonous or of a special character, much valuable data has been collected. In experimenting the rat was found in its varied diet and habitations to approximate most closely to man as an omnivorous feeder. Mixtures of substances, the components of which make up the animal body, including protein, a source of glucose which may be starch, cane sugar, malt sugar, milk sugar carefully purified, with the minerals sodium, potassium, calcium, magnesium, chlorine, iodine, phosphorus, sulphur and iron, fed to rats, were found to be insufficient for nutrition during development and adult life, without the addition of certain other substances, four or more in number, whose presence was only shown by the effect of their absence, the so-called vital food accessories or vitamins.

It is doubtful whether any recent research work has appealed more to the public fancy than the discovery of these vitamins, so much exploited commercially and held up as a panacea for many of the ills to which human flesh is heir. Even *Punch* chronicled their advent:

Vitamins, Oh! Vitamins
Ye vital sparks of eggs and beans.

Vitamins are not foods in the ordinary sense of the term as tissue builders or producers of energy. Their part is to aid the body to utilize food material sufficiently to get the cells to function. They are essential to growth and normal nutrition; their absence leads to disease and death. They are found in the germs and outside coverings of seeds such as wheat and rice and throughout the whole seed in peas and beans. They are abundant in the cells of yeast, the yolk of eggs, the green leaves of plants, the brain, liver and kidney of animals. A diet to maintain health must contain them in small but definite quantity and quality which as McCarrison shows "vary with age, sex, species and individual idiosyncrasy." Animals derive them directly from plants or fresh animal foods. Some are soluble in oils and are found in animal fats and fish oil. Butter from the milk of cows fed on green pastures prevents polyneuritis and œdema in pigeons, whereas that from the milk of cows dry fed does not. Without vitamins, proteins, carbohydrates, fats and salts are dead foods and cannot sustain life and growth.

Vitamins provide the cells of the body with the capacity to work, as is demonstrated by the recovery from certain conditions when such are added to the diet. Specific deprivation of any one of these vitamins which have already claimed a fifth of the alphabet in their designation, produces a specific syndrome which has been designated as a deficiency disease. These are xerophthalmia due to lack of the vitamin fat-soluble *A*, beri-beri, a multiple neuritis due to lack of water soluble *B*, scurvy due to lack of water soluble *C*, and rickets from lack of the fat-soluble vitamin *D*. Another fat-soluble substance recently discovered as essential to production is known as vitamin *E*.

As vitamin *B* has recently been shown to comprise two substances both essential to growth, the two fractions of the vitamin *B* complex are called vitamin *F* and *G* by a Committee of the American Public Health Association—*F* for the antineuritic substance associated with the name of Funk and *G* for the factor which Goldberger believes to be the preventive of pellagra. The latter has also been designated "P.P." or pellagra preventive, in contradistinction to the "B.P." or beri-beri preventive factor. A better nomenclature is "B1" (antineuritic) and "B2" (antipellagric).

The first observation of the inadequacy of a diet consisting solely of proteins, carbohydrates, fats, salts and water was made in 1888 by Lunin. From the effects of diets on mice he noted that "substances indispensable for nutrition must be present in milk besides casein, fat, lactose and salts."

In 1906 Gowland Hopkins showed that young rats declined and died on this purified food, but throve with the addition of à little milk to the diet.

In 1911 Mendel and Osborne, having shown that rats were unable to thrive on a diet of proteins, starch, sugar, lard, agar and inorganic salts, hinted the cause to be the lack of some essential element supplied by a small ration of milk.

In 1913 McCollum and Davis found that the ether-soluble fraction of butter and eggs supplied the missing factor, whilst lard and olive oil failed to

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 29, 1928.

do so. In 1915, investigating the dietary deficiencies of rice, they concluded that "there are necessary for normal nutrition during growth two classes of unknown accessory substances," which they termed "fat soluble *A*" and "water soluble *B*."

Funk meanwhile invented the term "vitamin" in connexion with his study of fat-soluble *C*.

Each vitamin is but a member of a team and the team itself but a part of a coordinated whole.

Colonel McCarrison refers to suitable protein, inorganic salts and vitamins as the essential constituents of a diet. An insufficient proportion of any one of these meant a lowered standard of physical efficiency, as was seen in man and his domestic animals in many parts of India. Minor manifestations of ill-health which escaped observation might follow, although as Hopkins said twenty years ago, they "affect the health of individuals to a degree most important to themselves." In animals this subnormal nutrition was shown by loss of "condition" and by impairment of fertility and of the power to rear their young. Where the deficiency was pronounced, distinct disease was produced in man, such as keratomalacia, night blindness, dental caries, polyneuritis, beri-beri, pellagra, scurvy, rickets, osteoporosis, slow healing of fractures, sterility, anæmia, some types of goître, alimentary dystrophy, gastric atony, diarrhœa, constipation, intestinal stasis, colitis, unhealthy skin, disordered action of the adrenal glands, stone in the bladder and vesical irritability.

McCarrison showed that vitamin defects lead to endocrine changes; the adrenals and pituitary enlarge, while other endocrine glands show more or less atrophy. Hence there must be a resultant imbalance. Pighini has found that if tadpoles are fed on thyreoid gland from pigeons in which beri-beri had been induced by an exclusive diet of polished rice, the tadpoles failed to show the acceleration of growth which is produced by administering the thyreoid gland from normal pigeons. This suggestive experiment points to the assumption that a deficiency of vitamin *B* has a definitely inhibitory effect on the healthy functioning of the thyreoid gland. For more than a century before the vitamin content of cod liver oil was discovered, the oil was used on account of its known beneficial effect in raising the patient's resistance, particularly to pulmonary affections.

Xerophthalmia—Vitamin A.

Fat-soluble *A*, discovered simultaneously by McCollom and Davis and by Osborne and Mendel in 1913, is essential for growth. Xerophthalmia, produced experimentally in rats, is first indicated by œdema of the eyelids and increased secretion in the eyes. Ulcers form on the cornea, but quickly disappear when butter fat, egg yolk, cod liver oil or fat from the intercellular fats of the glandular organs (liver, kidney, testicle) and the green leaves of plants are given. No vegetable fat or oil is effective, however. In Japan the condition is treated with chicken liver, in India by goat's liver, in Denmark by cream and fresh milk; in Roumania during the war the condition was cured by cod liver oil.

Beri-Beri—Vitamin B.

Beri-beri, a form of severe peripheral neuritis seen chiefly in polished rice-eating peoples, as in Japan, the Philippines and the East Indies, is not strictly a tropical disease, but occurs when the diet is deficient in the antineuritic vitamin *B*.

Eijkman in 1897, whilst medical officer of a prison in Java, where beri-beri cases were frequent, noticed that fowls fed on the refuse rice showed paralytic symptoms similar to his patients. Eijkman demonstrated that pigeons fed on polished rice developed polyneuritis, whereas those fed on unhulled rice did not. It was thought to be due to the loss of protein and phosphorus in the cortex.

Holst in 1900 disproved this by quoting an outbreak among Norwegian sailors with a diet rich in meat.

Foods were classified into those preventing beri-beri or containing vitamin *B* and those not preventing beri-beri or without vitamin *B*.

Foods not Containing Vitamin B.	*Foods Containing Vitamin B.*
White rice.	Whole rice (brown rice).
White flour.	Wholemeal flour.
Pearl barley.	Whole barley.
Sago.	Rye flour.
Tapioca.	Wheat germ, rice germ.
	Yeast, yeast extract.
	Peas, beans, nuts.

Beri-beri had a toll of 300 cases per 1,000 men in the Japanese Navy in 1870 to 1876. It is now free from the disease. Besides the loss of weight there is progressive weakness and loss of coordinating powers of the muscles with degeneration of the peripheral nerves.

McCarrison shows that with deficiency of vitamin *B* there are degenerative changes in the thymus, thyreoid, testes, spleen, ovary, pancreas, heart, liver, kidneys, stomach and intestines, whilst the adrenals suffered hypertrophy. He also shows that a deficiency of vitamin *B* makes animals and men susceptible to infections from pathogenic organisms.

In Labrador and Newfoundland, with a diet of meat, fish, molasses and tea, as in New Guinea, whenever the diet is restricted to bolted white flour degerminated corn meal and polished rice, beri-beri has appeared. This indicated the need for the eating of whole cereal grains, peas, beans, potatoes, milk, eggs, the glandular organs of animals or the green leaves of plants, such as lettuce, cabbage and Brussels sprouts.

Scurvy is a scourge which from the earliest records has played havoc with soldiers in camp, sailors, prisoners, pioneers and explorers. It was shown as early as 1734 by Backstrom to be "solely owing to a total abstinence from fresh vegetables and greens, which is alone the primary cause of the disease."

Dr. James Lind published a treatise on scurvy in 1756, embodying observations among sailors, in armies, in prisons and in besieged cities and among

the civil population of Russia. He showed that the deprivation of fresh uncooked vegetables caused scurvy to occur within a few weeks. On May 20, 1747, he took twelve patients with scurvy on board the *Salisbury* at sea. Two of the patients had two oranges and one lemon a day.

> The most sudden and visible good effects were procured from the use of oranges and lemons; one of those who had taken them being at the end of six days fit for duty. The other was the best recovered of any in his condition; and being now deemed pretty well, was appointed nurse to the rest of the sick.

The celebrations recently at the bicentenary of Captain Cook record how successfully he prevented scurvy on his voyages. The *Endeavour* left Plymouth on August 26, 1768. Twenty-seven days previous thereto the Lords of the Admiralty wrote to Lieutenant Cook:

> Whereas there is great reason to believe from what Dr. MacBride has recommended in his book entitled "Experimental Essays on the Scurvy, and Other Subjects" and his pamphlets entitled "An Historical Account of the New Method of Treating the Scurvy at Sea" (of which you will herewith receive copies) and from the opinion of other persons acquainted with scorbutic and other putrid diseases: and whereas we think fit experiments should be made of the good effects of it in your present intended voyage and have with that view directed the Commissioners of the Victualling to put a quantity on board the barque you command. There follow directions for the making of the infusion of wort and the prescription of a quart a day. The Surgeon is to keep an exact journal of the effects of the wort in scorbutic and other putrid diseases . . . noting down . . . the cases in which it is given, describing the several symptoms and relating the progress and effects from time to time, which journal is to be transmitted to us at the end of the voyage.

Assistant Surgeon Perry reported at the end of the voyage:

> Sauerkraut, mustard, vinegar, wheat, inspissated orange and lemon juice, saloup, portable soup, sugar, molasses, vegetables (at all times when they could be got), were, some in constant, others in occasional use. These were of such infinite service to the people in preserving them from scorbutic taint, that the use of the malt was (with respect to necessity) almost entirely precluded.
>
> We passed Cape Horn, all our men as free from scurvy as on our sailing from Plymouth.
>
> Three slight cases of scorbutic disorders occurred before arriving at Otaheite. Wort was given, with apparently good effect, and the symptoms disappeared.

To this it may be added that no opportunity was, as appears by the journal, ever lost of getting wild celery and any other wild herb that presented itself.

On his second voyage, after twenty months, on February 6, 1774, there was no scurvy and very little sickness of any kind, an indisputable proof of the untiring supervision Cook exercised over the health of his men.

In the last voyage of four years and two months it is recorded that not the slightest symptom of scurvy appeared in either the *Resolution* or the *Discovery*, so completely were Cook's precautions successful.

Captain Wharton speaks of Cook's greatest triumph as the suppression of scurvy. It is evident that it is to Cook's personal action the success was due. Wallis and Byron had antiscorbutics, but they suffered from scurvy; Furneaux, sailing with Cook in the second voyage under precisely similar circumstances, suffered from scurvy. It was only in Cook's ships and in the *Discovery*, commanded and officered by men who had sailed with Cook and seen his methods, that exemption occurred.

Contrast Cook's experience with that of Vasco de Gama who in 1497 in his voyage to the East Indies lost a hundred out of a hundred and sixty men from scurvy. No one who has not actually seen the ravages of this disease, can realize the terrible conditions caused thereby.

It was my painful privilege when serving in the Egyptian Quarantine Service, to witness the plight of the remnants of the Turkish garrison which had been besieged in Sana by the rebels in the Yemen, from which they were rescued by Feizi Pasha in 1906. The first relief column, so far from bringing success to the starving garrison, added to their distress, as before entering Sana, their transport was captured by the Bedouins. When the remnant of the garrison ultimately reached the coast and took ship at Hodeida, they presented a spectacle reminiscent of the sieges of ancient times. A German colleague, Dr. Vy, stationed at Suez, told me that over a hundred died before or after reaching Suez. In such a state of direful desolation was the ship that she went into voluntary quarantine and quite a large graveyard marks the spot at Moses Wells, the Suez Quarantine Station, where her freight was camped. It was the "Hakim Anglesy's" day on duty when the ship reached Port Said. I shall never forget the picture presented by the gaunt, haggard human skeletons, smitten with scurvy, their bodies covered with sores, their spongy gums bleeding, who could hardly crawl about the ship. One had died whilst passing through the Suez Canal. I was able to make that an excuse to keep the ship in quarantine and have quicklime and disinfectants sent on board before sending the ship to sea.

No doubt scurvy defeated Scott, Wilson and Oates in the Antarctic. Probably it was responsible to some extent for the condition of the survivors of the recent Nobile Arctic expedition. Recently a Norwegian barque was brought to Melbourne with the crew in a helpless condition, the result of the deprivation of fresh food.

Our modern knowledge of the cause of scurvy dates from the discovery of Holst and Fröhlich in 1912 that guinea-pigs speedily develope the disease when fed solely on cereals or bread. They showed that the antiscorbutic principle in fresh vegetables was destroyed by cooking or drying and that pulses acquired antiscorbutic principles when allowed to germinate. This fact was used when in Mesopotamia during the war by making peas and beans germinate before they were cooked.

Prevention.

Fresh citrus fruits and vegetables are the recognized preventives of scurvy. Germinated haricot beans and peas have been effective. In South Africa Kafir beer from fermented Kafir millet is effective in preventing and curing scurvy. Dried vegetables are useless.

Fresh meat has slight antiscorbutic properties as was proved by Stefanson in the Arctic.

Rickets—Vitamin D.

Although rickets had been recognized as a definite disease by Glisson in 1650 and Barlow in 1894 described the relationship between infantile scurvy and rickets, it was not until 1918, when Mellanby by feeding young puppies on diets which produced the disease, showed how it could be prevented by cod liver oil and demonstrated that it was a deficiency disease apparently associated with vitamin *A*. Previously rickets had been ascribed to various causes, such as intestinal intoxication, defective hygiene, disorders of the ductless glands or acidosis.

McCollom, Simmons, Shipley and Park severally showed that rickets could be produced in rats by diets poor in calcium or phosphorus or by diets either with a minimum of phosphorus and an excess of calcium or *vice versa*.

In 1916 Sherman and Pappenheimer showed that whilst rickets could be produced in rats on a diet low in phosphorus, it could be prevented by the addition of alkaline potassium phosphate and the withdrawal of an equivalent amount of calcium lactate.

Five days' feeding with cod liver oil was shown by McCollom and Simmons to cause the deposition of lime salts at the distal end of the femur and the proximal end of the left tibia, the so-called Shipley lime test showing a broad linear deposit which after staining with a 1% solution of silver nitrate and exposure to an arc light appears like a section of block honeycomb when examined by a binocular microscope.

McCollom, Simmons, Becker and Shipley proved that the antirhachitic substance in cod liver oil was distinct from the fat-soluble *A* by showing that this growth-promoting principle was killed by oxidation, whereas the antirhachitic agent designated vitamin *D* was not destroyed.

In 1912 Bosanyi demonstrated that the subcutaneous inoculation of aqueous extracts of normal bone marrow caused the healing of rhachitic bones in the rat. Extracts of bone marrow of rhachitic rats which had starved for five days and showed healing, were not antirhachitic.

In 1919 Holdschinsky cured rickets by exposure to the short invisible ultra-violet rays of a quartz mercury lamp.

Dr. Harriet Chick and Dr. Elsie Dalyell found that exposure to direct sunlight prevented and cured rickets in Viennese children.

Hess demonstrated a rise in inorganic phosphate in the blood of rhachitic children exposed to ultra-violet rays. It was found that cod liver oil had the same effect. Rickets was produced in rats by diets poor in phosphates and diets poor in calcium. With the addition of cod liver oil it was shown that the blood had a large calcium to phosphorus ratio in the one and a low calcium to phosphorus ratio in the other and that the structure of the bones depended on the concentration of the calcium and inorganic phosphorus in the blood.

Telfer found that extra calcium decreases the absorption of phosphorus in rats and infants.

Of interest to orthopædists is an experimental study in 1924 by Petersen of ununited fractures with regard to the inorganic bone-forming elements in the serum. By dietetic management he lowered the phosphorus content of the blood of dogs until the proportion of calcium and phosphorus was less than thirty. When bones of those dogs were fractured, they would not unite. When the phosphorus and calcium content were raised to normal, the fractured bones united.

Applying those principles to the treatment of ununited fractures with suitable additions to the hospital menu supplemented with cod liver oil or ultra-violet light, he found the fractures united as the progressive blood changes approximated to that of the normal adult.

In examination of employees of the Sydney City Council noting the comparatively rapid recovery of fractures in the case of men from the West of Ireland I ascribed this to the excellent condition of the bones of these people, reared largely on a diet of milk and bacon in a district where breast feeding is practically so universal that the infant mortality is even less than in New Zealand, where the mothers make a religion of the teachings of Sir Truby King as to regimen before and after child bearing.

No baby will ever have rickets and should have the foundation of good teeth laid if the mother during pregnancy and lactation has a diet with abundance of milk, eggs and green vegetables. The pregnant woman should eat spinach, celery tops, cabbage, turnip tops, lettuce. Fruits, carrots, turnips and beets as well as leafy vegetables prevent constipation and correct the defects of a diet composed mainly of bread, meats and potatoes. Cod liver oil is a preventive of rickets and effective in treatment.

Although Palm in 1890, studying the topography of rickets, considered that sunlight was a therapeutic agent and in 1904 Bucholz treated rickets successfully with artificial light, it is only recently that it has been proved that animals on a rhachitic-producing diet are protected against rickets when irradiated with ultra-violet rays and excrete fæces containing a substance which causes calcification in other rhachitic rats on reinoculation. The most remarkable fact, however, is that when natural foods non-rhachitic in character are irradiated they acquire antirhachitic properties. This is seen in the sawdust even of the cages in which rats are kept. Vitamin *D* is sparsely distributed in Nature, the best available source being egg yolk which is of great value in the dietary of the nursing infant. Chicken liver and fat are also of high protective value.

Hess found that cholesterol, when irradiated with ultra-violet light, acquired the power to induce calcification of the bones in rickets.

Ergosterol, a fractional product of cholesterol, has the same properties as the principle present in cod liver oil to which it owes its antirhachitic properties.

Research has shown that vitamin *D* is produced by ultra-violet irradiation of ergosterol, a derivative from cholesterol, a pro-vitamin. The effective rays in the formation of vitamin *D* are those occupying the middle portion of the ultra-violet spectrum (206-300). The shorter ultra-violet rays actually destroy vitamin *D* and should be screened off.

Peanut oil, other vegetable oils and olive oil become antirhachitic on irradiation. Ergosterol shows characteristic bands in its spectrum and yeast fats show absorption bands.

The reason why rickets is not a tropical disease is that the ultra-violet rays of the sun activate the cholesterol of the skin and some of the vitamin *D* is mobilized in the capillaries and transported throughout the body. The vitamin *D* of cod liver oil is a chemical substitute for sunlight. One can replace the other. This is why the women of Lewis in the Hebrides, who eat large quantities of fish oil, but do not get much sunlight, do not have children who suffer from rickets. This probably explains some of the remarkable results of the treatment of marasmic children by rubbing mutton bird oil on them, as recently reported in the lay press.

Vitamin E.

Evans and Bishop have published a series of observations on fertility and the presence of a substance in seeds and green leaves. Ether extracts of wheat germ or lettuce leaves give oils claimed to be efficacious in daily single drop administration. The vitamin *E* is fat-soluble, stable to heat, light and air.

FOOD DEFICIENCY DISEASES.[1]

By G. C. WILLCOCKS, O.B.E., M.C., M.B., Ch.M. (Sydney), M.R.C.P. (London),

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IT is my function to deal more particularly with the clinical aspect of food deficiency diseases and I propose to discuss these diseases shortly, as they concern us in Australia. Although general agreement has not been reached, rickets, scurvy, beri beri, infantile beri beri and pellagra are at present regarded as conditions due to defects in the food supply of the individual. Osteomalacia may also be classed under this head. It is a very common disorder in China and India where the diet is so different from ours. I am not sure if Barcoo rot which is still fairly common in our western districts, has been proved to be due to a vitamin deficiency, but I think most of us look upon it in that light to some extent. Boils and carbuncles are often regarded as evidence of diet defect.

Apart from vitamin deficiencies I am sure there is a great number of Australians who suffer from eating an ill-balanced diet, especially an excess of carbohydrates, but discussion on this question would I feel give us too much ground to cover to-night.

Pink Disease.

I would like to say a word about pink disease, because it appears to be more frequently recognised than formerly, and because food deficiency is still under consideration, I believe, as a cause. Erythrœdema, Swift's or pink disease as it is more generally known, is not uncommon in Sydney. Twenty-four children were admitted to the Royal Alexandra Hospital for Children with this diagnosis in 1927 and five died. In 1926 fifteen were admitted and six died. Pink disease occurs between the ages of five months and three and a half years. It is said to develope in previously healthy children, especially in the breast fed. It is characterized by fretfulness, photophobia, sleeplessness, stomatitis, sweating, loss of weight, a skin rash and cold pink feet. Hypotonia is a prominent feature. It lasts three or four months. Sudden death is not rare. The pathology is not well understood, congestion of the central nervous system, features of the hypoplastic lymphatic constitution, with bronchopneumonia and gastro-enteritis as sequelæ have been described by Warthin. In Sydney no constant pathology has yet been demonstrated. I understand the sympathetic nervous system is being especially examined, but no definite report has yet been made.

No method of treatment has given consistently good results. Ultra-violet light has its advocates, but in Sydney this treatment has not been particularly successful. Light clothing, fresh air day and night and fresh foods are recommended. Cod liver oil, calcium and lactic milk are also used.

The cause is unknown. Infection, toxins and food deficiency are suggested as possible factors. *Bacillus coli communis* infection is not uncommon.

Pellagra.

Pellagra according to Plimmer is due to a deficiency of vitamin B_2 or *PP* which is abundant in yeast and less so in wheat germ, eggs, dried peas, fish and tomatoes. Pellagra is common in Europe and the United States of America. Its main features are symmetrical erythema of hands, feet and face, followed by cracking of the skin; stomatitis and gastro-intestinal troubles are the rule and changes in the central nervous system are frequent.

It is a great rarity in Australia. I reported a case last year in which signs of pellagra and scurvy appeared to be combined.

Scurvy.

Scurvy we know as a rare disease of children. The signs of the advanced disease are pallor, anæmia, purpura and soft purple gums or stomatitis. Subperiosteal hæmorrhage gives rise to extreme irritability. I reported a case last year of typical

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 29, 1928.

clinical scurvy. The radiologists reported typical bone changes of rickets.

The diagnosis is often difficult and is often substantiated by the results of treatment with orange juice and fresh foods. Hess states that minor degrees of infantile scurvy occur frequently and I think it should always be borne in mind. It is due to a deficiency of vitamin *C*, the chemical nature of which is not yet known, but it is destroyed by drying, ageing and cooking of foods. It occurs in fresh fruits and vegetables. Fruit canned in the absence of air preserves its vitamin *C*.

Beri Beri.

Beri beri is due to the absence or deficiency of vitamin B_1, the formula of which is $C_6H_{10}ON_2$. It is contained in whole seeds, especially the germ of cereals. In Australia beri beri occurs mainly in Asiatics and the diagnosis is often in doubt. Peripheral neuritis with œdema is, however, a fairly typical picture. I reported a case in a European last year in which peripheral neuritis, anæmia, œdema and purpura occurred. This man had lived on bread and butter and tea for six months, because he had indigestion. A diet of meat, vegetables and orange juice cured him. I thought this was a mixed case of scurvy and beri beri and diagnosed avitaminosis, which term I think describes the condition found more accurately than a diagnosis of one entity. The three patients to whom I have alluded, all showed signs of defect of more than one vitamin.

Infantile beri beri has been reported in the Philippines, where it was carefully investigated, in Panama recently and last year in Nauru by Bray who wrote a treatise for his doctorate of medicine on the subject.

Nutritional œdema is described as a separate entity by some authors, but it may be that lack of assimilation of vitamin B_1 is the cause. In Lemnos in 1915 we had an outbreak of œdema with peripheral neuritis which I have regarded as beri beri. I do not know if that diagnosis has been corrected.

Plimmer states that the average diet containing white flour contains too little vitamin *B* and suggests using wholemeal flour or adding wheat germ or yeast extract to children's diets in clinics.

Rickets.

Rickets according to Thomson occurs in 50% of children in large British towns. Hess reports the same in New York. It is common in Sydney and apparently increasing. It is due to a deficiency of vitamin *D*, which is ergosterol activated by ultra-violet light. Two to four milligrammes of ergosterol are necessary daily for the health of a child. Ergosterol is extracted from ergot or yeast. The ultra-violet part of sunlight *plus* ergosterol is necessary to prevent rickets. Ultra-violet light does not pass through ordinary glass, nor through smoke nor dust. Hence the association of rickets with large towns and poor hygienic conditions. A definite quantity of ergosterol is necessary to prevent rickets; dilution of foods like milk with water, sugar, cereals or flour can therefore cause rickets by diminishing the amount of ergosterol ingested.

The symptoms and signs of rickets are familiar to all. As a rule the child does not thrive, will not sit up or walk, dentition is delayed, pallor and flabby muscles are noted, the head sweats and may be large or have a square shape. The forehead is often prominent and nodes may be detected on the ribs. Pot belly, Harrison's sulcus, bow legs and a tendency to bronchitis also occur. With these there is generally a history of unsatisfactory diet. The last two patients whom I have seen, were fed on "Glaxo" in too weak a dilution and for too long.

Constant reiteration is the only way of educating the public to the appreciation of the correct diet.

The treatment is well known. A litre of cow's milk a day, cod liver oil or radiosterol, fresh air and sunlight will cure most patients, if taken early enough. Egg yolk is a useful food in these cases.

The discovery that ergosterol is the necessary substance to prevent rickets was interesting. I quote Plimmer. At the Lister Institute in studying the effects of ultra-violet light as a preventive, it was found that rats did not get rickets if kept in cages which had been exposed to ultra-violet light. Other workers could not confirm this. It was then found that the rats in the original experiment had eaten the sawdust litter in the cages. Control rats in cages with non-irradiated sawdust got rickets. Evidently something in the sawdust became antirhachitic on exposure to the light. Various foods were then tested and it was found to be fat which became antirhachitic. The fats were narrowed down to cholesterol which was found to be capable of activation, but all cholesterol was not equally effective in preventing rickets.

Purification of cholesterol then showed that it contained very small quantities of an impurity; it was the impurity which was activated. This impurity was next identified with ergosterol. Finally pure ergosterol could be activated and minute quantities of irradiated ergosterol were able to cure and prevent rickets.

The bone change in rickets is softening, due to deficient calcium. There is also a deficiency of inorganic phosphates in the blood. The X rays reveal multiple joint affections with spreading out of the epiphyseal line and widening of the bone and the end of the bone has an inverted saucer-shaped appearance. Rickety rosary is a term applied to beading of the ribs, lines indicating condensed lime salts extend across the shaft. These are due to exacerbations of the disease.

Malnutrition and Marasmus.

Malnutrition and marasmus of children are also examples of unsatisfactory feeding due to a variety of causes. Frequent changes of diet are often at the bottom of these conditions. I believe *hydrargyrum cum creta* and the *pulvis hydrargyri*

cum creta compositus are very helpful in enabling these children to deal with the correct foods.

In my experience the subjects of these various ailments have rarely sought medical advice until the condition is advanced. I do not think the members of the medical profession as individuals are to blame for the frequency of these conditions. The solution lies in the education of the public, a difficult problem when you consider that the medical profession as a whole cannot reach the public who most need education.

Rheumatoid Arthritis.

I would like to mention an interesting study by Rowlands who found *Bacillus coli communis* infection in 80% of his patients with rheumatoid arthritis. In trying to ascertain how these organisms reached the bladder, he fed rats on a diet deficient in vitamin *B* which includes B_1 (anti beri beri) and B_2 (pellagra preventing), that is, on yeast, germ of seeds and animal protein foods. These rats developed desquamation of the mucosa and loss of the muscular coats of the intestine, with distention of the stomach and visceroptosis. As a result organisms entered the lacteal spaces of the intestine and invaded the body from the alimentary canal. Control rats fed on ample vitamin *B* showed no lesions of stomach or intestine and no organisms in the lacteal spaces. From this study Rowlands suggests that chronic rheumatoid conditions may be in part attributable to a diet deficient in vitamin *B*, causing an infection to spread from the bowel.

Acknowledgement.

I wish to express my thanks to Dr. H. R. Sear and to Dr. S. W. Ratcliff for the loan of films which I have shown tonight.

THE OCCURRENCE OF LEAD IN THE EGG OF THE DOMESTIC HEN.[1]

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PART II.

BEFORE proceeding to a study of the possible effects of the lead in eggs on irradiation with X rays after short periods of incubation, the work presented in this paper was undertaken. In this paper the results of lead estimations in eggs from various districts and a series of estimations on eggs from the same fowl are given, the method described in Part I of this work being used.(1) A note is given of the occurrence of lead in an emu egg and emu embryo and also some observations on the possible nature of the lead compound in eggs.

Briefly, the results here given indicate that lead occurs in all the eggs examined, collected from widely separated districts, and is independent of whether it is a "lead district" (as Broken Hill) or a country district, in which the lead might reasonably be expected to be of low concentration both in the air and ground. It is also shown that for eggs from the same fowl, the lead per egg is not constant, but varies within narrow limits for all parts of the egg.

Evidence is also produced which indicates that the lead compound in the yolk of eggs is probably a fatty acid compound, certainly not a diphosphate as has been postulated to occur in the blood stream.(2) Lead does not occur in detectable amounts in the blood of full-time hatching chicks.

Lead in Eggs from Scattered Districts.

To determine whether lead was present in all eggs or confined to those coming from "lead districts" where lead would be of common occurrence in the ground and so in the food of the fowl, eggs from scattered districts were examined. At first, the examination was confined to the yolks and the results for some of the eggs examined are given in Table I.

TABLE I.

Egg No.	Wet Weight of Yolk in Grammes.	Milligrammes of Lead found in	
		Whole Yolk.	100 Grammes of Wet Yolk.
1	21·3238	0·12	0·56
2	20·1316	0·105	0·53
3	21·2030	0·10	0·48
4	16·8592	0·10	0·595
5	16·2610	0·035	0·215
6	18·9646	0·075	0·395
7	19·0646	0·09	0·47
8	17·2750	0·055	0·32
9	14·4616	0·036	0·25
10	17·6346	0·065	0·37
11	13·8452	0·04	0·29
12	18·3466	0·045	0·24
13	14·6232	0·14	0·955
14	18·5498	0·14	0·76
15	16·9306	0·21	1·25
16	13·8148	0·17	1·2
17	16·2160	0·125	0·77
18	18·4282	0·270	1·45
19	19·3970	0·28	1·45
20	19·1422	0·19	0·99
21	17·1896	0·22	1·25
22	20·2414	0·29	1·40
23	24·4418	0·18	0·74
24	19·5282	0·07	0·35
25	21·7558	0·17	0·795
26	19·4768	0·21	1·08
27	18·2276	0·16	0·87
28	18·2356	0·12	0·66
29	21·0488	0·22	1·05
30	16·6416	0·27	1·63
31	20·0428	0·18	0·90
32	17·2130	0·28	1·62

Numerous eggs were examined for lead in shell, yolk and white and lead was always found to be present. Table II presents a representative distribution of lead in four eggs from widely separated districts.

The eggs so far examined were from domesticated fowls. It was thought that an analysis of some eggs from birds living under natural conditions would be of interest. Through the courtesy of Miss Winifrid Mankin, B.Sc., two emu eggs were collected from the Nyngan district, near the Bogan River. One of the eggs contained an approximately half-

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

TABLE II.

Egg No.	Nature of Material.	Wet Weight of Material in Grammes.	Milligrammes of Lead found in	
			Complete Part.	100 Grammes of Wet Material.
1	Shell	6·3456	0·160	2·58
	Yolk	14·1692	0·090	0·615
	White	30·3376	0·135	0·44
2	Shell	7·2646	0·22	3·02
	Yolk	19·5800	0·115	0·59
	White	34·7560	0·066	0·190
3	Shell	7·7100	0·230	3·00
	Yolk	17·0000	0·11	0·665
	White	32·1087	0·180	0·565
4	Shell	7·2065	0·13	1·82
	Yolk	17·4062	0·19	1·09
	White	31·4084	0·050	0·155

time emu chick: both eggs contained lead and an analysis of one of them is given in Table III.

TABLE III.

Part.	Wet Weight in Grammes.	Dry Weight in Grammes.	Percentage of Water.	Ash as Chloride.	Lead in Milligrammes found in	
					Part.	100 Milligrammes of Wet Material.
Embryo	121·90	19·2	83·6	1·67	0·025	0·02
Yolk ..	217·1	103·8	52·1	4·65	0·16	0·075
White..	64·8	10·7	83·5	1 25	0·17	0·025
Shell ..	91·7	89·14	2·8	—	0·68	0·74

Lead thus appears to be present in eggs whether from domesticated or wild birds and is absorbed by the embryo during growth. This point will be investigated in detail in a later paper.

Lead Content of Eggs from the Same Fowl.

The varying amounts of lead in eggs from different fowls in different districts made it of interest to

TABLE IV.

Total Weight in Grammes of Egg.	Fowl at Roseville (North Shore), Sydney.								
	Wet Weight in Grammes of			Milligrammes of Lead found in			Milligrammes of Lead per Hundred Grammes of Wet		
	Shell.	Yolk.	White.	Shell.	Yolk.	White.	Shell.	Yolk.	White.
56·4582	7·3182	15·8000	33·3400	0·12	0·14	0·13	1·64	0·885	0·390
52·8056	7·0848	14·2454	31·4754	0·09	0·145	0·145	1·28	1·0	0·46
53·8744	7·1164	14·5144	32·2436	0·065	0·135	0·14	0·91	0·93	0·435
53·1676	7·1586	15·7704	30·2386	0·07	0·135	0·135	1·0	0·855	0·445
55·9812	7·1074	16·8766	31·9972	0·11	0·14	0·12	1·55	0·835	0·38
53·3547	7·6357	16·2335	29·4855	0·08	0·14	0·13	1·05	0·865	0·44
56·0815	7·6650	17·0245	31·3920	0·10	0·15	0·14	1·30	0·88	0·445
56·3961	7·3865	16·9196	32·0900	0·10	0·15	0·14	1·36	0·89	0·435

The three last series of figures were obtained a fortnight later.

TABLE IV (*continued*).

Total Weight in Grammes of Egg.	Fowl at Petersham (Western Suburbs), Sydney.								
	Wet Weight in Grammes of			Milligrammes of Lead found in			Milligrammes of Lead per Hundred Grammes of Wet		
	Shell.	Yolk.	White.	Shell.	Yolk.	White.	Shell.	Yolk.	White.
56·4446	6·4760	16·5040	33·4646	0·06	0·08	0·05	0·925	0·485	0·15
56·3378	7·1430	16·0142	33·1806	0·06	0·08	0·055	0·84	0·50	0·165
63·4064	7·5636	18·1303	37·7125	0·06	0·08	0·05	0·795	0·44	0·130
56·6138	7·1328	17·3900	32·0910	0·06	0·085	0·045	0·845	0·49	0·140
57·0062	6·8690	16·6990	33·4382	0·06	0·085	0·04	0·87	0·51	0·120
61·2597	7·5436	17·9045	36·8116	0·06	0·085	0·045	0·80	0·475	0·120
56·2992	6·7382	16·7200	32·8410	0·055	0·085	0·045	0·82	0·51	0·135

TABLE IV (*continued*).

Total Weight in Grammes of Egg.	Fowl at Dulwich Hill (Western Suburbs), Sydney.								
	Wet Weight in Grammes of			Milligrammes of Lead found in			Milligrammes of Lead per Hundred Grammes of Wet		
	Shell.	Yolk.	White.	Shell.	Yolk.	White.	Shell.	Yolk.	White.
58·0495	7·0260	18·1785	32·8450	0·040	0·067	0·034	0·55	0·37	0·10
59·2595	6·7635	18·9360	33·5600	0·031	0·049	0·043	0·46	0·255	0·13
58·6964	6·7095	18·1227	33·8642	0·029	0·044	0·042	0·43	0·24	0·125
59·2355	6·8775	21·0760	31·2820	0·031	0·055	0·039	0·45	0·26	0·125
57·2405	6·7460	19·5280	30·9665	0·03	0·05	0·04	0·455	0·255	0·13
60·2265	7·1875	18·5550	34·4840	0·03	0·04	0·04	0·41	0·21	0·11
59·4321	7·1373	19·2702	33·0246	0·04	0·04	0·044	0·56	0·21	0·135
59·1836	6·8582	18·7054	33·6200	0·03	0·05	0·04	0·44	0·265	0·13

observe whether the lead content of eggs from any one fowl in one district was constant or variable. For this purpose eggs from the same fowl in any one district were examined daily over a period of several months, the districts being widely separated. The fowls were kept in individual pens and given the ordinary fowl-food. In Table IV are presented some of the results obtained.

From the three series of results given in Table IV from three different fowls, it is obvious that the lead content varies greatly for different fowls (as would be expected from results obtained from eggs chosen at random and presented in Tables I and II) and also that the lead content of eggs from the same fowl varies, although within fairly narrow limits. This difference is real and not due to errors of analysis, as has already been shown in Part I of this paper. The results given in the dissected form of Table IV for each part of the egg make the differences in each egg appear greater than they are when the total quantity of lead per egg is compared. When this comparison is made we have the following result, Table V.

TABLE V.

Egg No.	Total Weight in Grammes.	Total Lead in Milligrammes.
Roseville.		
1	56·4582	0·39
2	52·8056	0·38
3	52·8744	0·34
4	53·1676	0·34
5	55·9812	0·37
6	53·3457	0·35
7	56·0815	0·39
8	56·3961	0·39
Petersham.		
1	56·4446	0·19
2	56·3378	0·195
3	63·4064	0·19
4	56·6138	0·19
5	57·0062	0·185
6	61·2597	0·19
7	56·2992	0·185
Dulwich Hill.		
1	58·0495	0·145
2	59·2595	0·12
3	58·6964	0·11
4	59·2355	0·125
5	57·2405	0·12
6	60·2265	0·11
7	59·4321	0·12
8	59·1836	0·12

The lead variation in each series is small and appears to be independent of the weight of the egg. The variation is large enough, however, to warrant the conclusion that the lead content of eggs from the same fowl is not constant.

Examination of Tissue and Bone of Normal Fowls.

The fowl from which the eggs of series D (see Table V) were obtained, was killed and the tissue and bone examined for lead. This fowl lived under ordinary yard conditions, except that it did not receive food-scraps, but only bran, pollard, wheat, some bone meal and sea shell grit. It was hoped that some figures for a fowl from a lead area would be available, but these have not been obtainable. However, the figures for an egg from a fowl which died of plumbism at Broken Hill have been forwarded by Mr. R. Tannahill, B.Sc., A.A.C.I., who has been engaged in an investigation of the occurrences of lead in miners from Broken Hill under direction of the Commonwealth Government. The results are given in Table VI. Although the figures are given for only one fowl, several have been examined at different times and in each fowl lead has been found both in the egg and the body tissue and bones. It will be observed that there is slightly more lead in the eggs of the fowl from Broken Hill than that of the Dulwich Hill fowl. From figures obtained by Mr. Tannahill for the tissues of the Broken Hill fowl, the author can state that the lead difference in the two fowls is considerable, that of the Broken Hill fowl being much greater than that of the local fowl.

TABLE VI.

Part of Egg.	Lead Content in Milligrammes of Eggs received from		
	Broken Hill. (No. 1.)	Broken Hill. (No. 2.)	Dulwich Hill. (Table V.)
Shell	0·02	0·02	0·03 (average)
White	0·02	0·08	0·04
Yolk	0·07	0·06	0·05
TOTAL	0·11	0·16	0·12

TABLE VII.

Lead Content of Fowl which laid Dulwich Hill Eggs.

Part of Fowl.	Lead Content in Milligrammes per Hundred Grammes Wet Material.
Leg muscle	0·21
Breast muscle	0·19
Femur	0·94
Complete brain	0·17
Blood	less than 0·005
Ovaries	0·52

Normal Lead.

Whether lead is an essential constituent of tissues or not is still a debatable point; the work of the author shows that it is present in all the fowls examined and also in birds living under natural conditions, when they might reasonably be expected to be more free from accidental contamination than those kept under domestic conditions. No mention has been found in the literature on the occurrence of lead under normal conditions in tissues other than human. A review of the early workers on this material is beyond the scope of this work, but reference may be made to the review of Aub, Fairhall and others.[2]

The Lead Compound in Egg Yolk.

It is not proposed to give here in detail the chemical work and experiments involved in the attempts to isolate the lead compound from egg yolk, as this will be done in a paper dealing with the chemical aspect alone in another place. The following broad outline will give an understanding of the position as it is at present.

Because the amount of lead present in egg yolk is so minute, usually about 0·5 to 1·0 milligramme in one hundred grammes of egg yolk, it seemed impossible to separate the lead compound and to identify it definitely. While it cannot be said that this has been done, it can be stated that the lead compound has been definitely localized in one fraction of the egg yolk and that its identification is not far distant.

It is believed by Fairhall and his collaborators[2] that the lead probably circulates in the blood stream as colloidal lead phosphate and they discount the theory of Oliver[3] that it is present as the albuminate. The author agrees that it is probably true that lead, deposited in the skeleton, does exist there as the phosphate. That this is not so for the lead in egg yolk and probably not so for tissues in general has been proven by the following experiments.

Experiments.

Various liquids were tried to determine whether it was possible to extract the lead compound from egg yolk. When the following solutions were used which had been tested for lead previously, the lead was found to be extracted from the egg yolk: methyl alcohol, ethyl alcohol, acetone, ether, 5% hydrochloric acid, 5% nitric acid, petroleum ether. In each case over 80% of the lead present in the yolk was extracted. The extracts in the case of acetone contained over 95% of the lead. Fairhall[2] states that the lead compound in bone is not affected by dilute acids, because of the buffer action of the calcium phosphate; as this buffer action is not present in egg yolk, and as the lead is extracted by dilute acids, it appears that the lead is present in some form other than the phosphate.

To prove this point an amount of colloidal lead phosphate equivalent to the total amount of lead present in egg yolk was added to a yolk[4] and the extractions repeated. In no instance was the amount of lead extracted greater than before the addition of the phosphate, showing that this was not affected by the extracting fluids.

Fractionation.

Several of the extracts made in the foregoing manner with the use of ethyl alcohol and acetone were distilled under reduced pressure. The extracts were concentrated under reduced pressure, dried in a vacuum desiccator, and then distilled at 0·5 millimetres pressure with the aid of a Cenco "Hyvac" mechanical pump. Three fractions were collected and an analysis of the seen fraction yielded 82% of the total lead present in the yolk, the remainder being distributed between the third fraction and the residue. The second fraction was semisolid, pale yellow and almost odourless. Further experiments are being made with this fraction to observe whether the lead content distils in a constant ratio with the fraction, on repeated distillation.

Precipitation.

With the acetone extract from egg yolk, it was found to be possible to concentrate the lead in the cadmium chloride-lecithin compound, when the method given by Levene[5] was used.

To sum up the position as regards the lead compound in egg yolk, it is now known that the lead compound is extractable with various organic solvents and some dilute acids, confirmed from independent sources for human *post mortem* specimens as far as solubility in ethyl alcohol is concerned; also that the addition of colloidal lead phosphate to egg yolk does not lead to an increase in the lead extracted, showing that the compound present is probably not a phosphate.

The lead compound can be collected in one fraction by distillation of the extracted oil under reduced pressure, which would appear to exclude lead phosphate, some organic compound as oleate or glyceride being more probable.

The lead compound can be precipitated with the cadmium chloride-lecithin precipitate by means of Levene's method. While this does not exclude the possibility of a lead phosphate in the light of the two preceding paragraphs, it seems most probable that the lead is present as a compound of lecithin.

Lead and Lecithin.

Further experiments were made to observe whether there was any other chemical evidence to support the theory that the lead is present as a lecithin compound. It was found that ordinary commercial lecithin was completely precipitated by solutions of lead salts or colloidal lead, as prepared by Mr. R. K. Newman, B.Sc., for the Cancer Research Committee. A small sample of pure lecithin as prepared by Levene's method was not precipitated with lead acetate solution, nor with colloidal lead. The only apparent reason for the difference in these reactions is that the commercial lecithin contains the associated phosphatides such as kephalin and sphingomyelin. However, the fact that the lecithin is not precipitated as the lead compound, does not preclude the possibility of chemical combination.

The bearing of the lead phosphatide relationship to colloidal lead treatment of malignant cell growth has been dealt with in a paper by Blair Bell in *The Lancet* in 1924.

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SURGICAL IONIZATION APPLIED TO CYSTIC GOITRE.

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IT is a commonplace that new friends often blind us to the homely virtues of older and perhaps humbler comrades. The diathermy current has of late monopolized our attention to the exclusion of all other forms of electricity and the galvanic current has more or less fallen from grace. The galvanic current has also its victories and there is a wide domain of chemical and ionization effects in which no other therapeutic measure can compete.[1] It is, moreover, the fundamental current on which all other varieties of electro-therapeutics are based and an understanding of its effects and manipulation is a first charge essential upon every embryo disciple of the art. The newer currents, sinusoidal, Faradic, static, condenser and high frequency, are all mere variations of this current. It is therefore timely and fitting to draw attention to one of the most important properties of this current, the faculty of causing the migration of ions into the body tissues. This is generally known medically as ionization, a rather abused and confusing term.

Principles.

The physical principles underlying these applications[1] are simple, but must be thoroughly appreciated. The human body conducts electric currents because it is virtually an electrolytic solution containing an abundance of free ions. I shall therefore take as my starting point the passage of a current through a dilute solution of sodium chloride, the most abundant salt in the human frame. If two platinum electrodes be placed in such a solution, the pole by which the current enters, is known as the positive or anode and that by which it leaves, the negative or cathode. In any salt solution a portion of the salt is normally dissociated into its constituent ions, an ion being literally an uncombined atom *plus* an electric charge. The proportion of ions dissociated increases directly with the degree of dilution of the solution. One ion, the metal or basic ion, bears the positive charge and the other or acid ion, the minus; they are known respectively as the cation and anion. It is these ions which carry the current. Absolutely pure water or solutions of non-electrolytes, such as sugar, are non-conductors, since they contain no free ions. When current is turned on to the saline solution, on the principle that like charges repel and unlike charges attract, the positively charged sodium ion (cation) passes to the cathode (negative), whilst the negatively charged chlorine ion (anion) passes to the anode (positive). It will be noticed that the ion takes its name, not from the charge it bears, but from the pole to which it is attracted. Thus, the difference of potential applied to the two electrodes starts two streams of ions in opposite directions, anions from cathode to anode and cations from anode to cathode. On reaching their respective poles, these ions give up their charge and the aggregate of the charges given up constitutes the magnitude of the current in the circuit. The actual mass transferred is the product of current, time and a constant for the particular ion concerned known as the electro-chemical equivalent. Each ion has its own characteristic velocity and the conductivity of the solution depends not only on the number of ions present, but also upon the sum of the velocities of the two participating ionic charge bearers (Kolrausch's law). These velocities are minute and of the order of 10^{-4} centimetres per second; for example, the sodium ion has a velocity of 45×10^{-5} centimetres per second for a potential gradient of one volt per centimetre.[2] Accordingly, in practising surgical ionization, very deep effects from the ions *per se* would not be expected with factors of ordinary clinical magnitude.

Whilst holding the charge, the ordinary chemical affinities of the atom are satisfied, but on losing it at the pole these chemical properties return and the ion becomes the atom in a nascent and therefore very active condition. Consequently secondary reactions occur in the polar regions, the actual nature of which depends on the substance in solution and the composition of the electrodes. With sodium chloride and platinum electrodes the two following reactions occur;

(i) At the cathode:

$$2Na + 2H_2O = 2NaOH + H_2$$

(ii) At the anode:

$$2Cl + H_2O = 2HCl + O$$

The electrode being platinum, takes no part in the reaction. Two atoms of hydrogen are liberated at the cathode to one of oxygen at the anode, but if the electrode metal be attackable, the oxygen will oxidize the metal and fail to appear as a gas at all. In passing it may be mentioned that by plunging both electrodes in a weak saline solution, the electrode giving off the more gas may be confidently identified as the cathode. From these two equations it is clear that the reaction near the positive pole is acid and that near the negative is alkaline. Thus, if two platinum electrodes are plunged into living tissue, changes of this type occur and a caustic action, resulting in cellular destruction, takes place at both poles. But a further phenomenon becomes apparent. The current possesses the faculty of driving water molecules towards the cathode (cataphoresis) and that region becomes moister and softer, whilst the anodal portion waxes drier and harder. The positive needle will, therefore, tend to bind in the tissues, whereas the negative will be free in the presence of moisture and the soapy molecules of sodium hydroxide. Blood vessels will be constricted at the anode and dilated at the cathode.

It is physiologically proven[1] that the current has a sedative action near the anode (anelectrotonus) and an irritant and stimulatory effect near the cathode (catalectrotonus). When the current is increased slowly from zero, the first sensation felt by the patient is under the cathode; sometimes this is a handy reminder of wrongly connected poles.

It will be thus realized that metallic or basic ions must be driven in from the anode, since they are positively charged, and acid ions *vice versa*, from the cathode, since all metallic ions are cations and all acid ions anions. Some compound molecules, like cocaine and strychnine, also behave as cations and may be driven in from the anode.

The properties of the poles may now be tabulated as follows:

Anode.	*Cathode.*	
Acid	Alkaline	Alkaline froth
Liberates oxygen which attacks electrode	Liberates hydrogen	
Dry and binding	Moist and slippery	
Sedative and analgesic	Irritant and stimulating	
Vaso-constricting	Vaso-dilatating	
Drives in basic ions (cations)	Drives in acid ions (anions)	

The action at the poles is thus essentially a chemical caustic one which is intimately dispersed into the tissues in the immediate neighbourhood of the electrodes. The chemical energy, like the heat in diathermy, is produced by the current in the body tissues themselves, though, of course, only to a superficial extent.

Ionization for Medical Purposes.

Ionization, like diathermy, is divided into medical and surgical branches. Medical ionization is the introduction of selected ions by the galvanic current through the unbroken skin or mucous membrane to produce a local or general effect not of a destructive nature. Large pads soaked in a weak solution of the desired ion and of appropriate polarity are used as the electrodes. This part of the subject may be dismissed with the remark that its efficacy is in

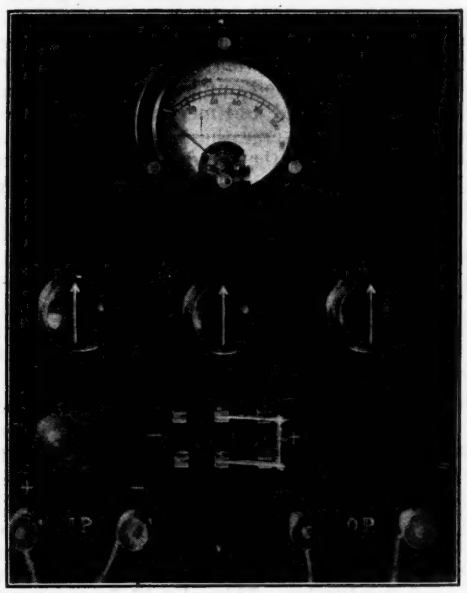

FIGURE I.
Lay-out of Control Board. O.P. = output; patient's terminals. I.P. = input, battery terminals.

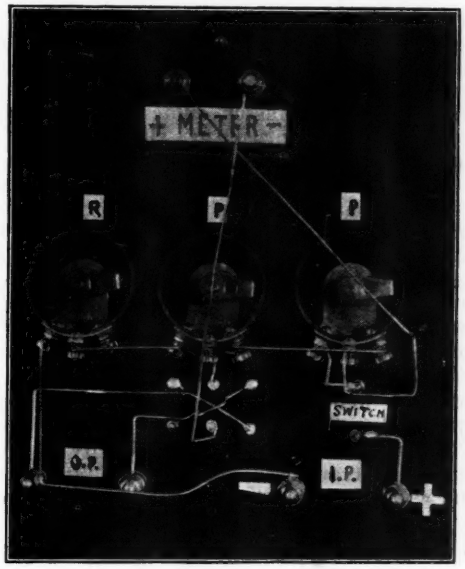

FIGURE II.
Reverse of Figure I, Showing Wiring. P. = Potentiometers; R. = rheostat in series; O.P. = output, patient's terminals; I.P. = input, battery terminals.

dispute[3] [4] as far as any deep action is concerned, though all are agreed that it is possible to introduce small amounts of drug ions through the integument into the immediate subcutaneous tissues and even into the blood stream.

Ionization as a Destructive Agent.

For surgical ionization the electrolytic and chemical actions at the poles are utilized to destroy or sterilize tissues. The energy and action are concentrated at the points of small electrodes or over small areas; usually the second electrode is a large indifferent pad soaked in saline solution which merely completes the circuit. There is, however, a second or bipolar method whereby both poles are inserted in the tissue to be destroyed when a destructive as distinct from a polar or ionic effect is desired. Massey[4] has developed this method to a high pitch. With an anode consisting of multiple zinc needles and a steel cathode common to all of them, both poles are inserted in the growth and up to four thousand milliampères of direct current are passed. The current literally coagulates the intervening tissues with a combination of heat and zinc ions. It is diathermy and ionization combined with the direct or galvanic current. It is, of course, impossible to pass such large currents unless they can be confined to the area to be eliminated. The more usual technique is with one active electrode of a polarity suited to the desired action.

The nature of the effect varies with the ion selected and in surgical treatment this is generally a metal which is applied from an anode fashioned as a plate, needle or rod of the selected substance. Zinc is a favourite and exerts a strong caustic influence probably by reaction with the hydrochloric

acid and nascent oxygen at the anode to form zinc oxychloride. Mercury, which may be amalgamated with other metals used as anodes, has a vigorous antiseptic action which lasts for some time in the devitalized area. Massey quotes a case where a large coagulum ionized by zinc and mercury remained sterile for twelve days after the operation. The copper ion is reputed to be hæmostatic and so on, each ion having its own characteristic effects. Occasionally solutions of ions are driven in from soaked pads for destructive effects, but the effect is said to be much milder. Some anions, notably chlorine, are thus used for the dissipation of fibrous tissue as in keloid, salicylic acid to sterilize infected ulcers, and iodine. In this case the active pole will, of course, be the cathode.

Surgical ionization as a whole is extremely adaptable in direction, dosage and control and a useful variety of effects in the diversity of ions is available. It removes diseased and adventitious tissue with a minimum of disfigurement and without opening the tissue spaces at any stage of the subsequent repair, thus rendering the possibility of infection and metastasis as remote as with the heat coagulation of diathermy. The last word on the use of this interesting physical agent has yet to be said.

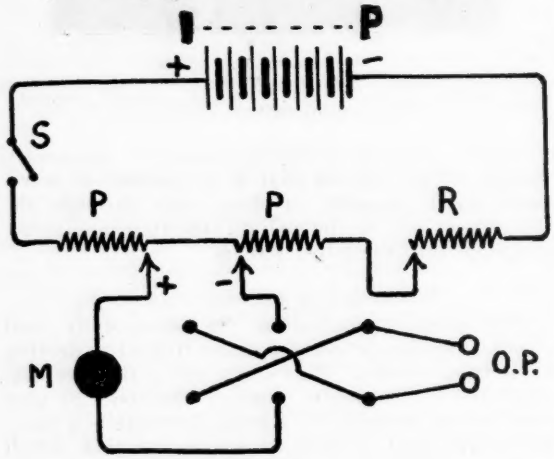

FIGURE III.

Diagram of Circuit. S. = switch; P. = potentiometer; R. = rheostat; I.P. = input; O.P. = output; M. = milliampèremeter.

Practice.

The application of the above principles to the treatment of cystic goître is an excellent example of the flexibility of electro-surgical methods in that the wall of a cyst is removed without mutilation of the containing organ. The technique is applicable only to goîtres of a cystic nature. It is advisable to aspirate the swelling with a fine hypodermic syringe to establish the diagnosis beyond doubt. The failure to obtain fluid contraindicates the treatment.

The procedure consists of two steps: (i) The formation by cathodic electrolysis of a fistula leading to the interior of the cyst and (ii) the progressive destruction of the wall by zinc-mercury ionization. First, identify the poles of the galvanic set. This may be done either by the "gassing" test as described or by placing both poles on a piece of wet litmus paper. The anode gives an acid reaction and the cathode an alkaline. Mark the cathode beyond chance of error.

With the patient recumbent, anæsthetize the skin over the most prominent portion of the swelling with "Novocain." Take a fairly thick needle from an aspirating set and connect after the usual aseptic precautions to the cathodic pole by winding the wire firmly around the base. Place an indifferent saline pad about ten centimetres by fifteen centimetres beneath the base of the patient's neck and connect to the other terminal, insuring that no metal touches the skin. See that all controls are at zero and puncture the cyst obliquely with the cathodic needle. The cyst will usually collapse with escape of the fluid contents, so that it is essential to fix the needle with its point inside the cyst by guying to the neck with a looped piece of strapping. Gradually turn on the current till at the end of two minutes the meter registers fifteen milliampères; leave it running for ten minutes. Some frothing will occur.

An error in connecting the aspirating needle to the wrong pole will result in driving the plating off the shaft and ferric ions into the skin, producing an indelible stain. After ten minutes slowly reduce to zero and remove the needle which will slip out easily, as it is a cathode. There remains a fistula with sealed edges leading to the interior of the cyst.

To render this permanent and to initiate the ionization of the cyst wall, replace the aspirating needle on the same lead with a needle of zinc cut from commercial 0·08 centimetre (one thirty-second of an inch) plate amalgamated with mercury. This amalgamation is carried out by dipping alternately in 30% sulphuric acid and mercury and then washing off the acid. Reverse the leads so that the zinc is now connected to the anode; insert deeply into the fistula and turn on the current gradually to fifteen milliampères for ten minutes as before. Zinc and mercury ions are now driven in and a whitish discoloration occurs in the neighbourhood of the needle extending outwards about one millimetre. No deep penetration into the thyreoid need be feared as the slow metallic ions soon give up their charge to speedier ions, combine locally and thus limit the action to the cyst wall.

This concludes the first sitting and henceforward every three or four days the zinc and mercury ionization is repeated without any local infiltration, though I have found it advantageous to place a drop of 10% cocaine solution on the wound. This is driven in by the anodic current and produces a fair anæsthesia, so that after a little time seven to ten milliampères will be tolerated if the current is increased gradually. This should be maintained for nine or ten minutes. On the third or fourth sitting the slough at the entrance of the fistula

due to ionization of the skin will come away and after that to limit its extension and the subsequent scar the zinc electrode is insulated where it touches the skin with a drop of sealing wax. After three weeks the interval between sittings may be extended to a week and the intensity of the current reduced, so that the final treatments from the fifth week onwards are of low intensity, merely insuring that the sinus heals up from the bottom. The tiny wound in my patient was healed completely in the eighth week, leaving an insignificant scar three millimetres across, while a small area of increased density replaced the cyst and the outline of the neck was restored to normal. The patient remained at work throughout and only complained of a "stiff neck" once or twice.

One or two points need mention. The zinc becomes very friable and brittle with the amalgamation, so that great care is necessary not to break it off in the sinus and at the same time the mercury ion is necessary to insure the sterility of the cavity. Never use the same electrode twice. In the early stages when the sinus is perhaps one and a half centimetres deep, the electrode being an anode, tends to stick and resists withdrawal in which case a reversal of the current, making it the cathode for one minute will loosen it, but the change must be made gradually and not by turning over the reversing switch with the current full on.

For a dressing of the wound between sittings I have used "Emollientine," but any bland antiseptic ointment will serve equally well.

The advantages of this treatment for a common condition are that it is positive, allows the patient to remain at work and inflicts a minimum of disfigurement. It substitutes a minor procedure for what appears to the patient to be a major surgical operation. The dangers are nil, as all tissue spaces are continuously sealed and infiltrated with antiseptic ions till healed.[4] Glandular hyperplasia is not benefited nor is it advisable to apply it in such cases. There is no apparent reason why cysts in other portions of the body should not be treated on the same lines after evacuation of their contents.

Apparatus.

Many practitioners are deterred from using the galvanic current by the high price of motor-generator sets, but much of the work can be performed with the following equipment which is made of radio components and costs only a few pounds. The source of power is two radio "B" dry batteries, forty-five volts each, connected in series to give ninety volts or better, but more expensive, one eighty-volt rechargeable "B" battery, really a battery of small lead accumulators. The quality of the current from a dry cell or accumulator is vastly better than any which can be produced by mechanical means and in this design there is full control of the voltage from zero to maximum. The construction of the set necessitates the following materials and they can be put together by anyone with an elementary knowledge of electricity.

One sheet of bakelite, twenty-three centimetres by twenty-five and a half centimetres (nine by ten inches), one milliampère meter (100 milliampères), three potentiometers (400 ohms), one double pole, double throw switch, one toggle switch, four binding posts, six lengths of light round busbar, two 45-volt radio "B" batteries and two and a half metres of light flex (for patient's leads).

All components should be suitable for mounting on the bakelite. Figure 1 shows the design of the board, Figure II the wiring at the reverse side and Figure III is a circuit diagram to indicate the principle of the control.

It will be noticed that the third potentiometer is used as a rheostat (one terminal being unconnected) and is in series with the resistances of the other two. The patient's circuit comes off from the central terminals of the two potentiometers marked P (Figure II) and thence through the meter to the central contacts of the double pole, double throw switch which is wired as a reverser and connected to the output or patient's binding posts (O.P.). Care is necessary in wiring to see that all the controls turn in a clockwise direction and for that reason the outside connexions of the right hand potentiometer, P (Figure II), are crossed.

The operation of the set is simple and consists in putting all controls to zero, switching on and successively rotating each of the dials from left to right (Figure I) until the desired intensity is reached, each dial being turned to its limit if necessary before proceeding to the next. The switch must be turned off at the conclusion of the treatment or the battery will be exhausted. In all the figures given the controls are at zero position.

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CONSIDERATION OF SOME OF THE LITERATURE AND PERSONAL EXPERIENCE IN THE TREATMENT OF GASTRO-ENTEROPTOSIS.[1]

By E. S. MEYERS, M.B. (Sydney),
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THE subject chosen for my paper tonight is one that does not always find favour in higher surgical circles and when the topic is introduced to such, it is liable to be received with about as much enthusiasm as a reference to the doings of the n'er-do-well of the family by some well-intentioned outsider. Nevertheless as the years go by, sponsors of excellent surgical repute are appearing to say a good word for this subject and I have no doubt that

[1] Read at a meeting of the Queensland Branch of the British Medical Association on November 2, 1928.

ultimately it will find an honoured place in our surgical text books.

Gastro-enteroptosis has not been a popular study with practitioners generally. It masquerades under a variety of names, splanchnoptosis, enteroptosis, gastro-coloptosis *et cetera* and there is no popular eponym which will suggest to the patient a single cause for his suffering. Not only in our time has this attitude towards medical problems been apparent, for in 1886 W. S. Playfair considered that the tendency of the advanced medicine of that day was unfortunately to overlook cure in the zeal for accurate diagnosis and correct pathology and to forget that patients come not to have their complaints diagnosed, but to be relieved of their suffering.

In presenting the subject to you I shall attempt to place before you all the factors that should be considered in order to give relief to our patients. Firstly, as to the anatomical aspect of the subject, my interest in this subject dates back to 1919 when I came across an article entitled "The Principles Underlying the Surgical Treatment of Gastro-intestinal Stasis Due to Causes Other Than Structural or Ulcerative Conditions," by R. C. Coffey.[1] Coffey has gone very thoroughly into the anatomical basis of the question and I shall attempt to bring out the main points he considered important by the use of the balopticon. Coffey makes great use of illustrations from Huntingdon's "Anatomy of the Peritoneum and Abdomen." The former states:

> In studying the abdominal cavity from the standpoint of comparative anatomy, we find that in man the liver has fused with the diaphragm, while in the quadruped it is suspended by a mesentery the same as other organs. In man the duodenum is firmly fixed to the right abdominal wall, while in the quadruped it is freely movable. In man the ascending and descending colon and the two flexures are normally fixed to the posterior abdominal wall without the intervention of mesentery, while in quadrupeds the large intestine has a long mesentery and is therefore freely movable. In man the great omentum grows down over the transverse colon and adheres to it. This does not occur in the quadruped. In man the omental bursa is usually obliterated by adhesion of its layers together. Obliteration does not take place in the quadruped. In man the pancreas has been rotated behind the peritoneum and fixed to the abdominal wall. In quadrupeds the pancreas lies between the layers of the mesentery.

"Why the difference between the animals walking on four legs and those walking erect?" Coffey believes these prenatal fixations by adhesion or fusion in the erect animal are without doubt for the purpose of holding the organs in their places, thus to prevent their piling up in the bottom of the abdominal cavity by gravity.

I shall throw on the screen pictures illustrating the process of rotation and fixation that occurs in the embryo. Now there are two points emerging from a study of Coffey's contribution to the literature that I wish to take up here. Coffey points out that anatomists have stated that in one out of every five human anatomical subjects the ascending colon has not properly fused with the parietal peritoneum. The second point is that according to Coffey the transverse colon has only feeble supporting mesenteries and this will be more pronounced where there is no obliteration of the omental bursa. Such a state of affairs will more easily allow of kinking and ptosis than when the transverse colon is better supported.

At this point I wish to consider the question from the physiological aspect. Many hold that the effect of the peritoneal adhesions is not of such great importance as has been claimed and that the absence of such fixation is of small significance. I think that J. Riddle Goffe has summarized the physiological aspect of the question most accurately in a definition that he has given of intraabdominal pressure. He defines intraabdominal pressure as the pressure within the abdomen due to external atmosphere pressure, to gravity, to muscular contraction of its wall and to intravisceral pressure. "The ligaments are the factors that suspend the organs and determine their fixed relative positions." In connexion with the abdominal wall (and with this must be considered the bony framework) I shall now show you a further picture taken from Coffey's article. While going very fully into the question of peritoneal adhesions, Coffey takes care to point out that most of the heavy organs are supported by other influences, namely, the shape of the abdominal cavity, the presence of fat and by the abdominal wall. The illustrations show how the organs are held on a shelf to a certain extent by the strength of the abdominal wall "much as if you should set a vessel on a shelf not quite wide enough to prevent it from falling off and should then nail a strap in front of it." The fatty packing in the abdominal cavity plays an important part in assisting to keep up the intraabdominal pressure and this fact is the basis of the so-called Weir Mitchell treatment by forced feeding.

Symptomatology.

According to Coffey the following steps occur in the production of general ptosis: (i) Deficient peritoneal fusion, (ii) sagging of certain portions of the alimentary tract with a consequent kinking at the fixed points, (iii) absorption of fat and letting down of all organs as a result of stasis.

At this point I want to refer to Waugh's contribution to the subject. In 1919 there appeared Waugh's article[2] on the consequences of a mobile ascending colon with a record of 180 operations. The results of the operations were most satisfactory.

Waugh states dogmatically that an ascending colon which preserves its primitive mesentery, is not a mechanism so mechanically efficient for its task as an ascending colon which has become securely fixed through the disappearance of its mesentery. He is of the opinion that the abnormal mobility of the ascending colon leads to inefficiency of function with failure of peristalsis. It thus becomes overloaded and drags on various organs. In this way a train of symptoms is produced varying according to the organ or organs dragged upon.

In 1928 Robert B. Carslaw,[3] of Glasgow, wrote an article entitled "Right Sided Visceroptosis."

Carslaw's observations on the subject are based on a series of 242 patients treated by right colopexy. He endorses very fully the claims of Waugh and he is of the opinion that in the majority of cases of general visceroptosis the primary condition is a right sided ptosis. In regard to the cause of failure of peristalsis referred to above, Carslaw thinks that this has been explained by Tyrrell Gray, who believes that the abnormal mobility of the colon results in a pull upon the mesentery with inhibition of the normal movements. This results in stasis and a vicious circle is set. In a breakdown of the normal abdominal supports many structures are likely to be affected, these will include nerves, vessels, viscera, ligaments *et cetera*. Hence it is no wonder that we have been presented with various clinical pictures. Clifford Albutt wrote in 1884 that "neuroses above the belt are more clearly understood than those below."

I wish now to present to you clinical pictures drawn by various authorities. Treves, writing in Albutt's "System of Medicine" in 1897, states:

> The results of general ptosis may be manifold and distressing. There are in the first place general asthenic symptoms of general depression and ill health. The patient becomes invalid and unfit for exertion. She is readily tired and is very liable to fainting and is only comfortable on lying down. There is a sense of weight in the abdomen and of a sickening dragging. Gastric symptoms are prominent. Conspicuous are a sense of burning in the epigastric region, vomiting, pain, loss of appetite and distress after food, colic is common.

Another clinical picture is that described graphically by A. E. Fossier[4] who states:

> Mental and physical fatigue are the predominant symptoms of hypotension. . . These patients tire easily. They cannot perform the day's work of their more fortunate brothers. They feel the need of mental and physical rest and the intensity of their symptoms is alleviated in the recumbent position. The extremities are usually cold, cyanosed, moist and clammy.

Is it fanciful to imagine that this is the history of an individual in whom there has been a breakdown of the mechanism necessary for a plantigrade existence?

Waugh's account of the general clinical characteristics associated with the presence of a mobile ascending colon is one that demands the fullest attention and I am sure that if you read Waugh's article, it will remind you of histories oft-times related. So faithful is the picture that you may recall the actual physiognomy of the fortunates (or unfortunates) who have crossed the threshold of your surgery. Waugh describes five types of cases, gastric, duodenal, biliary, renal and right iliac fossa according to the character and localization of the pain. Time does not permit a consideration of all these types, but I wish to give you Waugh's description of the general clinical characteristics which he says are common to them all at first. I cannot do better than quote Waugh's actual words:

> The onset is generally dated by the patients from the age of twenty years or thereabouts and described by them as the appearance of indigestion. There is no more than a feeling of fullness and discomfort after meals, flatulence and some lethargy. This indigestion cannot be explained either by indiscretions of diet, bad habit of life or unsuitable environment and it is not amenable to any change from one of the supposed causes.
>
> The average duration of these symptoms is about six months and then pain in one or other region of the abdomen appears for the first time.
>
> During these six months, many remedies have been tried without effect and with the appearance of pain, the patient is no longer content to regard the trouble as being of a trivial nature. Particular emphasis must be laid upon one aspect of pain in these cases. Although it is constant in type and constant in location for individual cases it is characteristically erratic and irregular in its times of appearance both with reference to the number of days on which it may be present followed by intervals of entirely irregular duration and to its appearance, disappearance and reappearance in the course of a single day.

I wish to throw on the screen here the after-history of three patients who had suffered from symptoms such as have been described. The history of the first is most interesting:

> I saw her first when she was on the operating table and a diagnosis of gall stones had been made, but she had had so many operations that the surgeon in charge refused to operate on the diagnosis made. This patient, M.J., *ætatis* twenty-seven, had had three operations without relief, she had been in hospital on two occasions after these operations and had been discharged as a case of neurasthenia. On the last occasion, as I have said, she was thought to be suffering from cholecystitis. I had recently read Waugh's article and the patient in describing her symptoms, repeated almost *verbatim* the story as told by Waugh. To cut a long story short, no gall stones were present, but the patient had an ascending colon with a very long mesentery along its whole length. Colopexy was performed three and a half years ago with excellent results.

So far the clinical pictures presented are of the earlier stages of the disability. Coffey's account of the later stages of the malady is a melancholy one. He writes:

> The organs settle towards the bottom of the abdomen, the upper abdomen shrinks in size to fit the organs contained in it. The chest follows and becomes elongated, the lower ribs become slanting and close together, while the lower abdomen becomes greatly expanded. Thus Nature, handicapped by congenital defects, gives up one stronghold after another, the last being the splenic flexure and retreats before her enemy, gravity, burning all her bridges behind her for the purpose of maintaining the one feature essential to life, *viz.*, an unobstructed alimentary canal.
>
> Such a patient, in an effort to make the best of things, assumes a characteristic attitude. The concavity of the back is gone. The shoulders lean far back to bring the centre of gravity directly over the legs. The hips and lower part of the spinal column are thrown well forward in an attempt to get the horizontal psoas line as near as possible under the prolapsed organs.

Time does not permit me to describe acquired visceroptosis consequent upon deficiency in the abdominal wall and pelvic floor following pregnancy, though I shall make further reference to the subject when I come to treatment.

Diagnosis.

A careful history and a thorough examination will go a long way in helping to arrive at a diagnosis. The most common mistake is a diagnosis of appendicitis when this organ is non-offending and much could be said of the "rape of the appendix."

One other pitfall must be mentioned, namely, failure to distinguish the hypochondriac from the

genuine sufferer and such a failure is one likely to bring the operative treatment into disrepute.

Treatment.

Treatment may be non-operative or operative and the former should nearly always precede the latter; in my opinion too much insistence cannot be placed upon this point, as in many cases it will be found that operative treatment will not be necessary. The restoration of normal function of the abdominal walls will in many cases be sufficient to restore our patients to health in spite of deficient peritoneal support.

I shall throw upon the screen pictures showing methods of mechanical supports used for such time as the patients are unable to rely on their own muscles. A further series of pictures will illustrate some of the operative methods in use.

Just a brief word as to prophylactic treatment, last but by no means least, and there is only one type of case I shall consider, namely, visceroptosis following child-birth. I think the greatest cause for this condition is the scant consideration given to the abdominal wall after pregnancy, due apparently to the lack of realization of the function of this important part of human anatomy in assisting to maintain the normal posture.

It is my practice in these cases to reeducate the overstretched abdominal and pelvic wall so that, by the time the mother resumes her household duties, she has something worth while to depend upon. I know this is not the usual practice, as many nurses think one a little strange in insisting on this routine, but they very soon realize the value of the treatment. Time does not permit me to go into detail, but I can assure you patients are grateful for the trouble taken.

Results.

I have treated many patients by non-operative and operative measures and the results have been most satisfactory. Patients should be submitted to operation only after mature consideration and I have no cause to be dissatisfied with the results obtained.

Conclusions.

In attempting to place before you my personal impressions of the subject there are many aspects that it has been impossible to mention in a short paper.

I have formed the opinion that, if care is taken in the selection of cases, operative treatment is very satisfactory and much invalidity and even death may be prevented. I know of two patients that I considered to have died from inanition as result of unrecognized and untreated visceroptosis.

There is much to be learnt from comparative anatomy and in this respect the work of Professor William Colin Mackenzie is known to you all and should receive every encouragement.

Too little is taught of the anatomy of the living. Hadley stated in his Listerian Oration at Adelaide in 1924:

> When student days are over those who carry out clinical work, are separated by too great a chasm from the physiologist and anatomist who gave us our first education.

As a profession we have not given sufficient attention to the question of physical training in health and disease, the work being left to the school teacher, drill sergeant, physical culture expert and masseur.

The school and industrial medical officer, the physician and surgeon in conjunction with the anatomist and physiologist should be the authorities on this subject. With sound methods of physical training in vogue in our schools, factories, hospitals and in everyday life, much invalidity of the nature I have described will be prevented.

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VARICOSE ULCER OF THE LEG.[1]

By A. W. SHUGG, M.B., B.S. (Melbourne), *Hobart.*

I MAKE no apologies for this paper, as I feel that many practitioners so commonly regard chronic ulcers of the leg as conditions to be avoided on account of the unusual difficulty in obtaining good results and the consequent natural lack of appreciation on the part of the patient. As a result many people resignedly suffer years of discomfort, pain and impaired efficiency.

This should not be, for Professor Unna gave us a method of treatment simple and efficient. The method is usually very sketchily described in text books, but its claims for more universal recognition were most ably advocated by Dr. Fay Maclure in THE MEDICAL JOURNAL OF AUSTRALIA.

I do not intend to dwell upon the ætiology or diagnosis of chronic ulcer of the leg. The three chief classes are: (i) Ulcers due to traumatism or to non-specific pyogenic bacteria, (ii) ulcers due to specific organisms, for example, syphilitic, (iii) malignant ulcers.

Any form of surface irritant may cause ulceration and all the factors predisposing to inflammation will hasten its occurrence: (i) Faulty nutrition, for example, long standing congestion of varicose veins, (ii) general debility, for example, that resulting from diabetes, (iii) anæsthesia of part of the leg, for example, in *tabes* or in section of a sensory

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on November 13, 1928.

nerve or nerves. These are all factors liable to further the ulceration process.

I propose to deal with one section only of the first class—varicose ulcer. I will not discuss the differential diagnosis, but wish to point out that varicose ulcer may occur in a person suffering from Bright's disease, diabetes, syphilis *et cetera* and then the general condition of the patient must be cared for in addition to local treatment.

Before describing the treatment I wish to recall to your minds the different stages through which an ulcer passes: (i) The stage of active ulceration or progressive spread, (ii) the transitional stage or that of balance or cleaning, (iii) the stage of healing and repair. The first and third stages run through definite clinical courses; the second stage may be quite short and transitory between the other two or it may be prolonged and complicated by certain accidental circumstances.

It is in this stage that the passive congestion of varicose veins causes delay in passing on to the third stage and according to local conditions the sub-varieties of "callous ulcer," "eczematous," "irritable ulcer" may occur. In addition to the congestion it is possible that other factors come into play in the non-healing of varicose ulcers. Most sufferers from this condition are middle-aged women, probably due to two incidents, pregnancy and its tendency to permanent varicosity of veins; parturition and perhaps an extension of a mild *post partum* streptococcal infection to the iliac and femoral lymphatic glands causing sufficient blockage to the lymph return to induce œdema of the legs on long standing or walking.

Treatment of the Ulcer.

First consider the case in which there is still ulceration going on; it is in whole or part belonging to stage one of an ulcer. It may be a recent ulceration or an old one that is spreading. There will be signs of inflammation, the surrounding tissues are œdematous and infiltrated and a considerable amount of pain will be present. The surface of the ulcer will be covered with greyish or dirty yellow material, partly consisting of slough, lymph and partly breaking-down tissues. Patients with this type should be put to bed with the leg slightly elevated for about ten days, when the inflammation and œdema will have considerably subsided and the ulcer be much cleaner.

The local treatment consists of frequent swabbing with equal parts of hydrogen peroxide and water and the application of hypertonic saline solution compresses. The former may sting and a weaker strength should be used. It is usually not advisable to prolong the use of the compresses.

This type of ulcer should then be ready for the, to my mind, most efficient method of treatment, Unna's paste. When the ulcer is first seen in a delayed second stage it is ready for treatment immediately. The essential in the success of the treatment by Unna's paste lies in the careful attention to technique.

If the leg is hairy it should be shaved two days before the first application and then swabbed twice daily with spirit. This prevents considerable pain when the paste is removed, as the hairs would be pulled out by the roots in the removal of the "stocking."

A one pound tin of paste is melted in a saucepan of water, care being taken that the water does not boil into the tin of paste. While the paste is heating, the ulcer is swabbed with dilute peroxide of hydrogen and the leg with spirit. A coating of the paste is then spread over the leg from the toes to just below the knee, including the ulcer. This may be done with a brush or an ointment knife, the latter for preference. A thin calico bandage is then applied, including the foot and ulcer and extending to just below the knee. Care must be taken to apply the bandage evenly and firmly, but not tightly. This bandage is then impregnated with the paste and another bandage applied in a similar manner. This in turn is impregnated with the paste and, the paste being allowed to a cool a little, a fairly thick layer is applied to the outside of the second bandage. When quite cold, the paste sets firmly, contracts slightly and forms a perfectly fitting "stocking." It is amazing and most gratifying the relief from pain and the comfort that these patients experience almost immediately.

The length of time during which the first stocking is allowed to remain, depends on the size and condition of the ulcer. The important sign calling for immediate removal and reapplication is the stocking becoming loose. From the outset, except in very severe cases, the patient is allowed to walk about and to follow his ordinary vocation, which he or she is usually able to do in perfect comfort. The stocking is removed by cutting up with a pair of scissors, care being taken not to snip the leg, and the stocking is pulled off. A considerable amount of discharge will have accumulated around the ulcer and has to be carefully cleaned away. This accumulation of discharge causes no discomfort and I am sure acts beneficially as an irritant to stimulate healing in the ulcer.

The leg having been cleansed and the skin swabbed with spirit, the paste is reapplied as already described. As healing progresses, the interval between applications lengthens gradually from one week to one month.

The advantages of this method of treatment of varicose ulcers are manifold and I wish to emphasize the four most important points.

1. The patients are able to follow their usual occupation after the first application in a great majority of cases.

2. The relief of pain is most pronounced and rapidly achieved.

3. The repaired ulcer is less likely to break down again than when healed by other methods (this point will be referred to again later).

4. The sureness of success if the technique is correct.

None of the subclasses of varicose ulcer, for example, "callous ulcer," "eczematous ulcer," "painful ulcer," fails to respond to treatment. Even the most thickened and adherent ulcer will with time and patience heal without recourse to any surgical measures.

Treatment of the Veins.

When the varicosity of the veins and the chronic congestion are not severe, it is surprising how rarely the ulcers recur. In the past it has been found advisable in all but the mildest cases to order *crêpe* bandages to be constantly worn. Proper directions for their application are necessary.

If the veins were severely varicosed, operation was recommended, but rarely accepted, the usual reason given being that the leg seemed so comfortable that the patient preferred to leave well alone. A certain number of these ulcers was bound to recur despite close attention to the proper bandaging of the legs. However, the introduction of artificial thrombosis of the veins by injections conferred a tremendous boon on these sufferers.

All patients after the healing of the ulcer should have their veins injected and thus the prevention of a recurrence should be assured. Small ulcers without a great degree of inflammation or congestion may be treated by injection without the application of Unna's paste. But if the ulcer is large, painful or inflamed, it is obviously advisable to heal or almost heal the ulcer before proceeding with the injections. I use quinine and urethane and believe the combination to be the most satisfactory of all the drugs used. The technique is simple and sloughing is most unlikely to occur if two rules are observed: (i) The needle should enter the skin at least 1·25 centimetres (half an inch) from the site of puncture of the vein, (ii) pressure should be kept up on the site of injection for some minutes after withdrawal of the needle.

Case Reports.

I wish to show two patients to illustrate my remarks.

CASE I. Mrs. O., *ætatis* fifty-nine years, had ulcers on both legs for twenty-three years; they appeared soon after birth of last child. There were two ulcers on each leg. On the right leg both were fifteen centimetres (six inches) in length, five centimetres (two inches) in width and about nine millimetres (three-eighths of an inch) deep situated in the lower third of the leg and the inner one adherent to the tibia. On the left leg the ulcers were similarly situated, smaller in size, but as deep. The legs were very œdematous and inflamed and extensive varicose veins were present in both legs. The pain had been severe for years and the patient rarely had an undisturbed night's sleep.

Treatment began on August 18, 1927, and after the first application of paste the pain practically ceased. After the second application the patient could attend to light household duties in comfort. By December 15, 1927, all the ulcers were healed. Early in 1928 one ulcer broke down, necessitating several further applications of paste. At the same time the veins in the other leg were treated by injections and when the other leg was healed, its veins were similarly treated. Now both legs give not the slightest trouble.

CASE II. Mr. T., *ætatis* forty-three years, a farmer, had three ulcers over the tibia in the lower third of the leg, each about the size of a florin, present for twelve months; he had increasing difficulty in carrying on with his work owing to pain. Varicose veins were rather prominent. Treatment began on May 26, 1928. After June 13 he was able to carry on with his work, ploughing in comfort and with no detriment to the healing process which was completed by the end of July. He was advised to have the veins treated, but so far this has not been done.

Reviews.

A MONOGRAPH ON THE STREPTOCOCCI.

PERHAPS no other family or group of bacteria has proved so difficult of orderly subdivision as the streptococci. The great Pasteur was one of the first to recognize these organisms. It is said of him that when at the Faculty of Medicine in Paris a physician spoke at length of the mysterious influences in puerperal fever, the Father of Bacteriology jumped up, contradicted the physician and drew on a blackboard organisms forming chains.

The streptococci were first isolated in pure culture by Fehleisen in 1883 and though much work has been done on them in these forty-five years and the number of differential tests and classification schemes has been legion, yet it cannot be said that even today a truly satisfactory subdivision exists. David Thomson, of London, has compiled a large volume on the subject.[1]

No less than 251 pages are devoted to an historical survey of the streptococccci and the author sometimes adds his own criticism. Then follow about twenty pages written by Dr. Warren Crowe, of Bath, a well known investigator in this field. Dr. Thomson devotes only about ten pages to his own methods and classification tables. There are, however, fifty-seven plates each usually containing six photomicrographs. He begins with a reprint of Crowe's illustrations from the latter's "Bacteriology and Surgery of Chronic Arthritis and Rheumatism." The main feature of the book, of course, is the photographic record of the various types met with. There are photographs of both colonies and stained films. The author bases his classification upon that of Dr. Crowe and this depends largely upon the use of similar media, namely, varieties of heated blood agar. The streptococci may not cause any alteration in the colour of these media or they may very slightly or moderately (green) or considerably (yellow) bleach them according to the amounts of hydrogen peroxide produced or they may blacken the media. Dr. Warren Crowe builds up four main groups, A, B, C, D, and Dr. Thomson five, A, B, C, D and E, and each has several subgroups. The result is certainly not simple or easily remembered and Dr. Thomson considers that the number of species is very large. Systematic classifications by different workers, using different criteria, are certainly of value, but most bacteriologists will still use specific terms such as *pyogenes*, *viridans*, until something of practical value can be gained from a large number of tests. In practice, several varieties of streptococci are nearly always present in material for examination from the mucous membranes of man. Therefore the bacteriologist usually does a primary plating and he has a choice of fresh blood agar such as that of J. H. Brown or of the heated blood agar of Warren Crowe or Thomson, and the classification of subcultures on these media will vary accordingly.

Thomson considers that the heated blood media are preferable. The photographic differentiation of colonies may prove useful more particularly to special workers, but like the serological results and fermentation tests with streptococci the complexity is rather overwhelming. To the general reader certainly the best part of the monograph is the historical review of the subject. The author is to be congratulated upon an admirable presentation of a laborious piece of work and a lengthy bibliography is recommended to those interested in this group of microorganisms.

[1] "Annals of the Pickett-Thomson Research Laboratory," Volume III; 1927. London: Baillière, Tindall and Cox. Demy 4to., pp. 320, with illustrations. Price: 42s. net.

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A Retrospect.

Oto-Rhino-Laryngology.

No important discovery has been made during the past twelve months in the physiology of the organs of hearing, of smell or of phonation. A few years ago some very valuable additions to the knowledge of the physics of the ear mechanism attracted attention. It was anticipated that as a result of the better understanding of the functions of the middle and internal ear, otologists would be able to learn a great deal concerning the diseases of these organs and to apply this knowledge to the prevention or cure of these diseases. Unfortunately this has not been realized. Nevertheless much steady work has been conducted and some progress has to be registered. In the United States of America a national campaign against oto-sclerosis was commenced two years ago, without any signal success. During the year some important discussions have taken place in England. It may be said that the disease is now better understood and that the prospects of gaining a mastery over the ætiological factors are becoming brighter. Hodgson has studied the bone changes by radiology and after much painstaking research he has evolved a technique for the radiological examination of the labyrinth by means of which the finest details can be clearly recognized. He has found that there is no true sclerosis in so-called oto-sclerosis; rarefaction of the cochlear portion of the labyrinth is noted. Later the rarefaction spreads to the vestibular area. Further investigation is required before the condition can be adequately defined.

Friesner and Rosen state that as a result of chemical studies of the pus issuing from the tympanic cavity, the calcium content is greater when the bone is involved than when the inflammatory process is simply a suppuration of the mucosa. They therefore recommend a mastoid operation whenever the calcium content of the pus is high. Dan. Mackenzie has advocated early drainage of the mastoid antrum in middle ear disease to obviate the occurrence of chronic suppuration.

Kent Hughes has again urged the use of diathermy in various forms of deafness. He has not discussed the pathology of the conditions benefited by the treatment nor has he given information concerning the way in which the diathermy acts on the altered tissues. That heat applied in an intense and convenient form is capable of producing reactive changes to inflammatory lesions, is well known. Whether the diathermic current acts more specially than does heat in other forms has yet to be demonstrated.

In the chapter of rhinology there is less to record. Improved results of treatment of nasal polypi have been reported from America as the result of radium applications. It is further stated that recurrence is retarded by radium treatment and that at times the removal by this agent is not followed by any recurrence.

Campbell has evolved a new technique for ionization of the nose and the nasal sinuses. The patient lies face downwards with the head lower than the trunk. The head is extended. The ionizing solution is allowed to run into the cavities and the nostrils are plugged. The author claims that he obtains better results from this treatment in sinusitis and in hay fever than from other forms of treatment, but he admits that the results in atrophic rhinitis are disappointing. He has also described his technique for repairing congenital palatal clefts by utilizing cartilage from the nasal septum.

Ozœna still causes much difficulty and trouble to rhinologists. Freudenfall and Stein have used rubber sponge packs with benefit. It is not clear how such packs act. Lamothe exhibits large doses of sodium salicylate internally and claims to obtain benefit therefrom. It is reported that diphtheria antitoxin has failed to yield good results, notwithstanding the enthusiasm manifested when the method was first introduced.

The treatment of malignant disease of the upper air passages seems to occupy the mind of laryngologists. Howarth has modified the technique of

the operation of laryngo-fissure. He does not remove the ala of the thyreoid cartilage; the tissues are stripped from the cartilage without damage to the cartilage itself. Neither does he recommend a preliminary tracheotomy. Colledge prefers laryngectomy and lateral pharyngectomy for cancer of the larynx, particularly for extrinsic lesions. He maintains that surgical removal is the only satisfactory method known at present and that treatment by X rays is both useless and dangerous. Watt and Finzi hold the opinion that radium is an effective method of treatment of endothelioma of the mouth and upper air passages and deep X rays yield the best results in sarcoma. They advocate short wave X rays for epithelioma. The primary tumours can be destroyed by radiation therapy, but the glandular deposits are more difficult to treat. Even in inoperable growths prolongation of life is frequently attained. They plead for greater coordination between the laryngologist and the radiologist and for the early reference of the patient to the two in consultation. Schmiegelow finds that for early intrinsic carcinoma of the larynx diathermy applied with great thoroughness and the complete destruction of all the tissues involved yield better results than total laryngectomy. He employs the same means for neoplasms of the nose, accessory sinuses, mouth and jaws.

Mandelbaum has invented a trochar for performing reverse tracheotomy. The trochar is passed through the mouth under the guidance of the sense of touch and through the larynx. It is forced between the tracheal rings through the wall and skin. He claims that this method is superior and quicker than the usual method.

Pædiatrics.

Some important investigations have been carried out during the past twelve months or more that will have the effect of enhancing the understanding of certain diseases of children. Among them are some concerned with metabolic processes. Toverud found that the carbohydrate tolerance is dependent on many factors, the most important of which are the severity of the diabetes, the control of the diet and the incidence of infection. He endeavoured to gauge the tolerance by ascertaining the amount of "Insulin" necessary to reduce the sugar content of the blood during fasting to the normal level. He has formed the opinion that an acidosis occurs during many of the infections and that this increases the disturbance of the carbohydrate metabolism. It is suggested that the acidosis leads to an increased glycogenolytic action of the liver and the muscles. In substantiation of his hypothesis he points out that all the diabetic children under his care who were kept free from infection, and whose diet was controlled, had an unchanged sugar content of the blood without any increase in the amount of "Insulin" given. He standardizes the diet of diabetic children on the basis of their carbohydrate tolerance. The amount of protein given is kept at three grammes per kilogram body weight for children under seven years of age and two grammes per kilogram for children over seven.

Agassiz and Gill have studied a condition in children which is frequently mistaken for pulmonary tuberculosis. It is a non-tuberculous fibrosis. The adventitious sounds in the chest vary considerably. During pyrexial attacks the sounds tend to become moister and more profuse. Bronchopneumonia is rarely present. The amount of oxygen taken up by the blood is diminished. In cyanotic children this diminution is very pronounced. The explanation given of the finding that the volume percentage of oxygen in the blood in fibrosis is lowered, while that in pulmonary tuberculosis is usually normal or even raised, is as follows: The fibrous process involves the alveolar walls and thus prevents the aeration of the blood. The blood circulates through the capillaries of the affected alveoli. Lundsgaard and Van Slyke have named this condition an unaerated shunt. In pulmonary tuberculosis the vessels in the deposit become thrombosed and in consequence the capillary circulation is completely arrested. When a child with fibrosis is infected and the process becomes tuberculous, the alveolar capillaries are thrombosed and the unaerated shunt ceases. This is manifested by the diminishing cyanosis as the disease progresses.

Reuben and Claman have collected a considerable amount of information from the literature concerning the chronic type of thrombocytopœnic *purpura*

hæmorrhagica. The operation of splenectomy appears to be a life-saving one. The blood platelets are deficient in this condition; after the removal of the spleen they increase in number at first. Later there is a fall, followed by a return to a normal or subnormal level. The hæmorrhages cease, the blood clots normally and the other signs of the disease disappear. The authors postulate a toxin which is formed in the spleen. They believe that this toxin increases the thrombolytic power of the spleen, causes the megakaryocytes to produce fewer platelets, affects the quality of the platelets and alters the Rouget cells, so that the capillaries become more permeable. The hypothesis is plausible and stands as a good working basis.

Reuben has pointed out that a close relationship exists between the incidence of valvular disease of the heart and the persistence of muscular fibres and blood vessels in the valves. In the fœtus endocarditis is more frequently encountered on the right side of the heart. The valves in this part of the heart are more vascular than on the left side. The muscle fibres persist longer in the aortic cusp of the mitral valve than in any of the other cusps and endocarditis of this cusp is commoner in adults than in children.

Some interesting observations have been made in regard to calcium and phosphorus metabolism in rickets and other diseases. Murdoch has shown that the absorption of phosphorus is the same in normal children as in rhachitic children. It is increased in children with healing rickets. The addition of calcium to the diet induces a fall in the phosphorus content of the blood. This was found to be greater in rhachitic than in normal children. The excess of calcium found in the fæces of rhachitic children is held to be due to the defective absorption of ingested calcium. The phosphorus is combined with the calcium to form an insoluble phosphate. Thus an excess of calcium, whether caused by increased intake or by defective absorption is equivalent to a diminished supply of phosphorus. There appears to be no doubt that calcium deficiency occurs in rickets. Holt demonstrated that excess of fat in the diet deceased the calcium absorption. Excess of fat leads to the formation of insoluble fatty acid soaps containing calcium. Calcium retention may be increased by the simultaneous use of activators such as cod liver oil or ultra-violet rays; the absorption is favoured by the presence of lactose. Calcium salts can be absorbed if given into an empty stomach, when the alkaline reaction of the duodenal contents is reduced.

Litchfield found that the calcium content of the blood of tuberculous children is deficient and that improvement followed the exhibition of full doses of calcium preparations. Calcium chloride and calcium lactate are usually employed. The dose may be reduced if the patients are exposed to sunshine or to ultra-violet rays.

Fries has shown that ultra-violet radiation does not alter the basal metabolic rate by more than 10%, a variation which falls well within the limit of error. He holds that the beneficial action of sunlight or ultra-violet rays is due to the reduction of the acid bodies of the blood. A rise in the blood alkalinity favours calcium retention.

Strong has written on the results of immunization against scarlatina. A toxin derived from *Streptococcus hæmolyticus,* prepared according to Dick, is employed. Five injections are given at intervals of from five to seven days. He maintains that the toxin should never be given to children who have been exposed to infection within a short time. It is not to be given to children with scarlet fever. Good results are said to have been obtained by the use of detoxified toxin, which retains its antigenic power.

In Australia some sound work has been accomplished. Gastro-intestinal affections have occupied the attention of several clinicians, and bacteriologists. E. H. M. Stephen has discussed ileo-colitis, gastro-enteritis and the chronic vomiting of infants in a very practical manner. Spalding Laurie has given valuable advice on the basis of his considerable experience in connexion with the treatment of the diarrhœas of infants. Lillie has published a preliminary note on the life-saving expedient of blood transfusion for infants with severe gastro-enteritis. Reginald Webster has dealt with spirochætal bronchiectasis, congenital heart lesions and some intracranial tumours in infants. The ætiology of pyelitis of infancy has been challenged by I.

Robertson who gives adequate reasons for a revision of the accepted teaching and for a fuller study of the mode of infection in this condition.

Orthopædic Surgery.

The record for the year 1928 of work carried out in orthopædic surgery is a relatively long one, but it contains little that is new. In Australia many subjects have been discussed and some eminently useful reviews of current doctrines have been given. Lockyer Potter has devised a new operation to remove the disability resulting from ulnar paralysis. He found that the deformity of the hand is caused by the hyperextension of metacarpo-phalangeal joints of the little and ring fingers and flexion at the interdigital joints. Careful observation led him to recognize that the fundamental defect in this condition is a loss of stability of the metacarpo-phalangeal joints. The operation is a simple one. He utilized portion of the paralysed tendon of the *flexor profundus digitorum* as a sling around the proximal phalanx, while the distal free end of the divided tendon is carried round the middle phalanx and across the proximal interphalangeal joint. As the joint is now steadied, the superficial flexor is enabled to provide movement.

In regard to the treatment of tuberculosis of the spine Hibbs and Risser have reviewed the results obtained from the operation of fusion. A large series of patients had been treated in this manner and close on 75% had been restored to health. They find that the best results are obtained in children. Early diagnosis and early treatment are essential. Their inquiries have taught them that after fusion for tuberculous disease of the spine, tuberculosis of other joints tends to improve in at least one-half of the patients. This experience is at variance with the common doctrine that fusion is apt to disseminate the disease. Spontaneous fusion is never complete and in consequence it is unwise to rely on such a termination. When there is paraplegia from above it is necessary to perform laminectomy. It is interesting to note that Rollier still maintains that the treatment of this condition should be conservative.

Congenital dislocation of the hip joint has again been the subject of inquiry and discussion. Melbourne orthopædic surgeons prefer conservative methods unless the patient can be subjected to operation before the age of eight years if one hip is affected or before the age of six if both are involved. They have obtained good results in older children with the aid of plaster casing or extension splints. Allison has also advocated early operation. He has stated that in many patients when arthritis supervenes years later after closed reduction of congenital dislocations, this is referable to injury to the articular cartilage at the time of the reduction.

An important discussion on acute osteomyelitis took place early in the year at the Royal Society of Medicine. The same subject was discussed by orthopædic surgeons later in the year at a meeting of the New South Wales Branch of the British Medical Association. Investigations have elicited the fact that the most common infecting agent is the *Staphylococcus aureus*. It is essential whenever unexplained fever is present in a child who has some affection of a long bone, to make a bacteriological examination of the blood. The path of infection has been fully investigated and the manner in which the inflammatory changes reach the medulla, has been subjected to careful analysis. It is held that the greatest care should be exercised at operation, for it is often impossible to ascertain how much of the bone may recover. Adequate drainage is imperative. Orr claims that the wound should be packed with gauze soaked in vaseline and the limb encased in plaster. He leaves the limb alone for from two to six weeks, even when the wound is foul. By this method he obtains complete rest and he is satisfied that the results are excellent. In regard to early operation there is some difference of opinion concerning the justification of opening the bone, unless the signs of definite osteomyelitis are present.

The prevention of paralysis in poliomyelitis is being regarded as an urgent problem. Many aspects of this subject were presented to the readers of this journal in a symposium on poliomyelitis. The essential point is early diagnosis.

Although nothing new has been published in connexion with the treatment of fracture, an article by C. Craig on the results he has obtained by adopting

Hamilton Russell's methods demands the attention of surgeons. It is becoming more generally recognized that excellent functional results are often obtained notwithstanding imperfections in the anatomical position. Skiagrams often exaggerate the deformity. It is doubtful whether complete reliance on skiagrams is justified, especially when the correctness of the treatment is the subject of inquiry in a court of law.

Dermatology.

Progress in science does not necessarily depend on the discovery of something new. Often the advance is first registered when a demand is made for the recognition of a half forgotten doctrine or of an unaccepted hypothesis. In the world of dermatology the most important event of 1928 is the energetic plea put forward by Herman Lawrence for a concerted campaign directed against the epithelial triad. In 1927 Molesworth put forward his ingenious contention that rodent ulcer and epithelioma of the skin of the face and lip resulted from the direct action of sunlight and especially of the ultra-violet rays contained therein. He emphasized the relative rarity of lupus in Australia; the rays of the sun exert a bactericidal action on the tubercle bacilli in the skin. Lawrence agrees with the greater number of Molesworth's observations, but he differs from him in regard to the ætiological complex that leads to keratoses, rodent ulcer and epitheliomata of the skin in Australia. He is disinclined to accord these changes to the chemical or physical effects of the sun's rays, but maintains that the association of much sunshine with relatively low humidity is the responsible agent. Whether Lawrence or Molesworth is right need not detain us at present; it is probable that an authoritative judgement may be delivered on this matter in the near future. The actual significance of Herman Lawrence's energetic movement and of Molesworth's powerful argument is that attention has been focused on this vital subject and many imperfectly recognized facts have now been definitely recorded. They have placed a fuller understanding of the epithelial triad within reach and have pointed out the way in which prophylaxis can be attained.

Norman Paul has described a form of pellagrous dermatitis in which congestion, inflammation, thickening and pigmentation and atrophic thinning are recognizable. The lesion is a hyperkeratosis; beneath the epidermis plasma cells and lymphocytes are accumulated in an inflammatory mass.

Paul has also described a pyogenic dermatitis with a verrucose appearance. Pus exuded through cribriform openings. He suggests that there is an acute infection of the skin caused by a streptococcus. Numerous minute abscesses form within the inflammatory plaque, which give rise to a large amount of destruction. Hyperplastic tags of epidermis form between the cribriform openings and this lends to the lesion its verrucose appearance.

Macleod and Dowling have studied the seborrhœic dermatitis caused by Sabouraud's bottle bacillus or pityrosporon of Malassez. The condition has been reproduced experimentally and its pathogenesis appears to be established.

Some interesting work has been conducted in connexion with congenital keratoses associated with bullæ and also in connexion with *ichthyosis bullosæ*. Goldsmith has drawn a sharp distinction between congenital ichthyosiform erythrodermia and *ichthyosis vulgaris*. He is satisfied that the latter is never associated with the appearance of bullæ. In the former condition the bullosis and the keratosis are distinct. Macleod has put forward some argument of favour of the development of bullæ from secondary infection in the presence of true ichthyosis. As an alternative explanation he suggests that the condition is an atypical form of *epidermolysis bullosæ*.

Eller and Bucky have carried out some observations on the action of rays lying midway between ultra-violet and X rays. They call these rays *Grenzstrahlen* or borderline rays. They claim that exposure within reasonable limits does not induce atrophy or telangiectasis. They give rise to a mild erythema in twelve to twenty-four hours. The rays have but little penetrating power and are absorbed by the skin. The action has been found to be absorptive. Many dermatoses are controlled by them.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Staphylococcal Meningitis.

F. H. LAMB (*Archives of Pædiatrics*, May, 1928) reports the history of an infant with staphylococcal meningitis who was treated by intraventricular injections of gentian violet. The infant was admitted with a large head (47·75 centimetres in circumference), bulging fontanelle, twitching of extremities, bilateral inconstant internal strabismus, increased tendon reflexes and spasticity of the muscles of the abdomen and extremities. Hydrocephalus was diagnosed. By ventricular, cistern and spinal puncture it was ascertained that a staphylococcal meningitis was present. The symptoms of acute meningitis appeared within a few days and for the first eight days treatment was general with the exception of one spinal puncture. Twelve days after the onset of the acute symptoms seven centimetres of 0·1% solution of gentian violet were injected into the lateral ventricle. Four days later the injection was repeated. After another four days lumbar puncture yielded a clear fluid under normal pressure with a cell count of 105. Culture yielded only a scattered growth of staphylococci. During the next month there was a great improvement and the acute meningitic symptoms subsided. The right parietal bone became depressed to such an extent that it was overridden by the margins of the frontal, occipital and left parietal bones. At the end of the month ventricular puncture yielded a canary-yellow, clear fluid with a cell count of 15; it was sterile on culture. The spinal fluid was bloody but sterile on culture. The patient continued to improve and the head diminished in size. The author considers the condition to have been one of obstructive hydrocephalus due to a low grade staphylococcal meningitis. The changes in pressure due to the first ventricular and spinal punctures probably released the obstruction and at the same time caused a spread of the staphylococci with a consequent acute inflammatory reaction. This was successfully treated by the intraventricular injection of gentian violet.

Tuberculosis in Children.

A. LEVINSON (*Archives of Pediatrics*, July, 1928) has studied the condition in a number of children under twelve years of age suffering from active tuberculosis. In one year he noted that 7·61% of the children admitted to the Pediatric Department of the Cook County Hospital, Chicago, were tuberculous. The main ætiological factor was contact. One or both parents of the majority of the children or some close relative suffered from tuberculosis. Measles appeared to be a predisposing cause in half the children in the series. In regard to race, colour and economic conditions there was a great preponderance of coloured over white children, possibly owing in part to the fact that they were living under less hygienic conditions. In the majority the children were under five years of age. Sixty-five out of the 119 children studied were suffering from miliary tuberculosis in which category the author includes tuberculous meningitis. The early signs and symptoms were vague. Many coughed, but in only a few was the cough distressing. Some had fine crackling *râles* and in some were found scattered areas of dulness. In the greater number, however, the diagnosis was made exclusively from the X ray findings. The author points out that tuberculous meningitis is always part of a generalized miliary tuberculosis and that most, but not all patients with a miliary tuberculosis sooner or later develope a meningitis. Apart from the miliary type, the lungs were affected in thirty other patients. Fifteen of these had acute and fifteen chronic pulmonary involvement. The most frequent physical findings in the acute condition were dulness, bronchial breathing and crackling *râles*. The symptoms were strikingly mild, the most common being night sweats and loss of weight. Nine of the series had tuberculous peritonitis, six the plastic form and three the ascitic form. The most frequent symptoms of the plastic form were severe pains, alternating diarrhœa and constipation and sometimes vomiting. The sufferers from the ascitic form rarely had pain. Three of the children had pleurisy with effusion. Many had palpable cervical glands, but very few had large masses. Apparently the patients in whom there was pulmonary involvement, had definite tracheo-bronchial adenitis. Amongst the series were also six patients with tuberculoma of the brain, two with Pott's disease, three with hip disease, one with pectoral abscess and one with pyelonephritis and cystitis. All of these also had tuberculosis elsewhere. The most useful diagnostic tests were radiological examination and examination of the sputum, blood and cerebrospinal fluid. The miliary disease would not have been diagnosed in many of the children but for the snowflake appearance of the chest on the X ray plate. In only a few patients were tubercle bacilli discovered in the sputum or stomach contents. Since children usually have a high leucocyte count, the absence of leucocytosis in the presence of pulmonary disease suggests tuberculosis. The author believes that by a persistent and careful search it is possible to find tubercle bacilli in the cerebrospinal fluid of many patients suffering from tuberculous meningitis.

Epidemic Encephalitis.

M. M. ANDERSON (*Archives of Disease in Childhood*, April, 1928) during the last three years has investigated eighty-three patients suffering from epidemic encephalitis who were seen at the Royal Hospital for Sick Children, Glasgow, from 1918 to 1927. Twelve of these died and eight cannot be traced, while sixty-three are known to the author at the present time (August, 1927). The picture was usually typical. It was not always possible to make a diagnosis in the acute stage, but later developments usually made it clear. Sudden onset was present in seventy-one instances and the most frequent early symptoms were insomnia in 50%, choreiform restlessness in 29%, lethargy in 26%, diplopia in 20·5%, headache in 15% and pain in 14%; vomiting and convulsions occurred with much less frequency. The period of acute illness lasted usually from two days to two months and fever was present for the first week or two. During this stage such symptoms as respiratory disturbances, myoclonus and ocular disturbances were often found. The acute stage was followed by an apparent return to health which lasted from a few days to a month. Later symptoms then appeared in the following order of frequency: Mental changes, nocturnal excitement, conduct changes, respiratory disturbances, Parkinsonian syndrome, loss of accommodation, choreiform restlessness, myoclonic movements and obesity. Lethargy was present in fifty of the eighty-three patients and was the first symptom noticed in twenty-six. It usually lasted from ten to twenty-three days and varied in depth in different patients. Choreiform restlessness lasted in most of its victims for from four to fourteen days and in most of them was followed by lethargy. This manifestation was found also in the later stages of the disease, but was not of such an extreme nature. Myoclonus was present in six of the patients, four of whom were in the acute stage. The facial muscles were affected in three, the diaphragm and extraordinary muscles of respiration in the other three. Of the seventy-three children seen in the later stages in this series, twenty-two had developed some degree of Parkinsonism. The period between the acute illness and the onset of this syndrome varied between a few weeks and three years or more. Immobility of the face or one limb was usually the earliest sign and this was followed in most instances by the characteristic facies, stance and gait. In the six most pronounced examples *talipes equino-varus* was noted; it was bilateral in four. Coarse or fine tremor was present in eight of the patients and excessive salivation in all but two. The greater number of those showing the most severe results had already passed through periods of bad behaviour, cruelty and filthy habits. The author notes the close resemblance between progressive lenticular degeneration and that of some of her examples. With regard to prognosis, only three out of the twenty-two children have shown any improvement, while in the others the syndrome has either remained stationary or has steadily progressed. There have been no deaths. Ocular paralyses were

present in twelve of forty patients seen in the acute stage. These were mostly transient, with the exception of paralysis of accommodation which showed a striking tendency to persist. Diplopia was present in nineteen patients and in all of them was followed by a period of excitement. Strabismus was found in eleven, ptosis in eight and nystagmus in six. Eighteen of the patients manifested loss of accommodation with retention of the reflex to light. In all except two of these the condition persisted. The most frequent paralysis seen after that of the eye was that of the seventh cranial nerve. This occurred in seven patients and was always transient. In six patients there were transient paralyses of the limbs.

ORTHOPÆDIC SURGERY.

Spinal Fusion Operation.

RUSSELL A. HIBBS AND J. C. RISSER (*Journal of Bone and Joint Surgery*, October, 1928) review results of fusion operation performed on 286 children with tuberculosis of the spine from 1915 to 1920 inclusive. Of these 74·6% were cured and 26·2% died, the majority of deaths being due to tuberculosis or directly related causes. The operative mortality was 9%. The patients in the main were of the poorer classes, coming from unfavourable surroundings and after operation returning to the same environment. He emphasizes the need of early diagnosis and early operation. After considering this series he concludes that the results offer abundant proof that the operation is more suited to children than to adults. In 50% of the patients tuberculosis in other joints was improved after spinal fusion without other treatment. This leads him to the belief that operations on tuberculous joints are not likely to disseminate disease. Paralysis in tuberculous spondylitis is always due to the products of inflammation pressing on the cord. Laminectomy in these circumstances must be done early and should be combined with fusion. Sinuses complicating the disease always increase its seriousness. When natural fusion takes place, it is always incomplete. This fact and the enforced inactivity over long periods is in his opinion a convincing argument in favour of radical treatment as the most favourable method in dealing with tuberculous spondylitis in children.

Myofascitis.

F. H. ALBEE (*The Journal of the American Medical Association*, November 3, 1928) reviews the pathological conditions underlying 275 cases of myofascitis, a term he applies to a chronic or subacute inflammation of muscular attachments to bone. He thinks that the various forms of fibrositis hitherto recognized under the terms lumbago, tennis elbow, sacro-iliac strain, flat foot are all local signs of a general toxæmia which arises from the bowel in 90% and from other foci in only 10%. He finds the Gram-negative flora in the intestine greatly reduced in number and the Gram-positive flora accordingly increased. Histamine is generally present in a large amount and mucus in the stool and constipation usually correspond with its presence. The stool is frequently acid to all indicators. He deprecates the frequent use of the term sacro-iliac strain or sacro-iliac subluxation to indicate a condition which he now considers to be entirely due to myofascitis. He states that he had many patients referred to him for operative fixation in whom nothing more than radical treatment for focal infection was needed to complete a cure. He outlines the methods of treatment of which the main principles are medicated lavage of the colon for ten days followed by implantation of living cultures of *bacillus coli*. He states that it is useless to treat local orthopædic symptoms unless the primary toxic condition is eradicated.

Cartilage Injuries.

WILLIS LASHER (*Journal of Bone and Joint Surgery*, July, 1928) describes injuries to cartilages and fibrous tissue in the upper limb. The areas of fibrous tissue located at the lateral surfaces of the phalanges communicate directly with the hyaline cartilage covering the bone ends and frequently contain cartilage cells which extend along the shaft beyond the limits of the finger joints. An injury of this fibrous area causes an unsightly deformity and slight flexion of the joint. Vigorous massage, provided it is given early, will prevent a great deal of the permanent deformity which is otherwise liable to ensue.

Sacro-iliac Subluxation.

EDWARD A. RICH (*Journal of Bone and Joint Surgery*, July, 1928) stabilizes the lower end of the spine in sacro-iliac subluxation due to undue mobility of the sacro-iliac articulations by means of a shot gun bone graft of about seventeen to eighteen centimetres in length implanted in the spinous processes of the second or third lumbar vertebræ and into the external lateral crest of the sacrum crossing the sacro-iliac articulations. The graft is split in the longitudinal direction to conform to spinal concavity. He claims slight ankylosis without sequelæ. When it is desirable to secure firm ankylosis of the sacro-iliac articulations he injects five to fifteen drops of supersaturated tincture of iodine directly into the articulation.

Malunion of the Femur.

MUIR BRADBURN (*New Orleans Medical and Surgical Journal*, April, 1928) describes the principles underlying the treatment of fractures of the femur with particular attention to malunion. Skeletal traction, properly employed, will overcome shortening in 100% of patients and this is the chief means of preventing malunion. When malunion has occurred, the fracture should be mobilized by open operation followed by gradual skeletal traction, as there is a tendency for malunion to follow mobilization by the closed method. Operations in which an attempt is made to correct great degrees of shortening after malunited femoral fractures, are usually attended with great shock and high mortality.

Legg-Perthes's Disease.

R. WHITMAN (*Journal of Bone and Joint Surgery*, July, 1928) describes the pathology of Legg-Perthes's disease as a primary necrosis of the epiphysis, caused apparently by defective blood supply followed by secondary absorption of the dead bone and its replacement by fibrous tissue and consequent structural deformity. The articular cartilage is largely destroyed, but in spite of the distortion retains its vitality. He considers there is no anatomical or pathological correspondence between this condition and the epiphyseal fracture or displacement; the nature of the process seems to support Legg's conclusion, namely that the final result is but little influenced by the character of the treatment. The degree of final disability is dependent on the character of the deformity and the secondary changes that it induces in the articulation.

Fracture of the Femur.

H. WINNETT ORR (*Surgery, Gynecology and Obstetrics*, August, 1928) describes his method for the treatment of fracture deformities of the femur. Provided it is a late fracture he does not hesitate to refracture a malunited femur by the open or closed method. The lower fragments may be controlled by ice tongs or by moleskin adhesive traction for maintaining extension; whichever method is used, the extension apparatus is fixed in a plaster of Paris casing for four to six weeks. The bone graft is usually not necessary for good union and good alignment is obtained in practically all patients treated by this method. In compound fractures the maintenance of the bone in correct alignment and with correct length should outweigh the considerations of wound treatment.

Congenital Pseudarthrosis.

M. S. HENDERSON (*Journal of Bone and Joint Surgery*, July, 1928) states that the consensus of opinion is that the cause of congenital pseudarthrosis is a local and not a general one. This conclusion has been arrived at after the study of a number of calcium phosphate readings. The general health needs attention and the patient should have a walking caliper (in the case of pseudarthrosis in the lower limb before operation) so that the lines of weight-bearing are as nearly normal as possible. Deformities should be corrected at operation and massage, heliotherapy, contrast baths, cod liver oil and vitamin-rich food are all adjuvant. Endocrine preparations have been used only empirically.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on November 29, 1928, DR. J. E. V. BARLING, the President, in the chair.

Food Deficiency Diseases.

DR. J. S. PURDY read a paper entitled: "Food Deficiency Diseases: Their Cause and Prevention" (see page 90).

DR. G. C. WILLCOCKS read a paper entitled: "Food Deficiency Diseases" (see page 94).

DR. HARVEY SUTTON said that as in so many other subjects, the progress of knowledge seemed to have enlarged the bounds of ignorance. Not only had vitamins *D* and *E* been added to the original grouping of *A*, *B* and *C*, but considerable discussion had occurred as to the exact relationship in both causation and treatment of various deficiency diseases of such factors as mineral salts, infection, sunlight and actinic rays, exercise, balanced diet and good quality protein. A further factor which had not received so much attention, but was inevitably concerned, was that of the internal secretions. The exact relationship of infection was a particularly difficult problem. How far might infection be causative and if causative, how far might it merely intensify the changes or be the real origin of the condition or was the infection not causative but merely an accidental association? For example, in pellagra the resistance to tuberculosis was remarkably affected and a very large number of Turkish prisoners in the Palestine campaign, affected by pellagra, had succumbed to a very rapidly fatal form of respiratory tuberculosis. On the other hand, in rickets infection played a much more important part in the actual causation of the disease, intensifying, if it did not actually create the processes concerned in the disease. Nothing was more characteristic of the history of rhachitic children than the early bowel disturbances, constipation alternating with diarrhœa and very definite intestinal distension, the so-called "pot belly."

It was difficult to know just what the relationship between an infection and a vitamin was in this early stage of rickets. While deprivation of calcium might cause bone changes they were considered to be distinct from the true rhachitic phenomena. A proportion of calcium and phosphorus in the food would seem to be helpful in its prevention. But presuming that this intestinal infection occurred, was it that the vitamin deficiency rendered the bowel more susceptible, while it at the same time caused changes in the bone and nervous system or was the absorption of poison from the bowel the next step in the production of the disease or did both vitamin and alimentary toxins cooperate before the disease occurred?

Still further complicating the question were the definite but not widely recognized prodromal symptoms and signs pointed out by McCarrison, where indigestion and bowel infection, various vague disturbances and infections of very varied type were prone to occur in the stage during which the deficiency in vitamins had not yet been sufficient to produce the classic characters of the fully developed disease. The occurrence of prodromal symptoms was one which should be much more widely appreciated both in hospital and in private practice.

In recent literature on beri beri similar discussions had occurred as to the question of infection or vitamin as the real causative factor.

With regard to relationship to internal secretions he felt that this aspect was one to which insufficient attention had been drawn. In spite of the welter of literature, often of most extraordinary type, dealing with endocrines, where language was used worthy of the prospectus of an oil company, the importance of relationship to vitamins was seldom mentioned. In some way or other these two factors, one external, one internal, decided the growth of the human body. It seemed not unlikely that they cooperated in some very definite way which did not yet appear to have been recognized. It seemed to him that they could not consider any question of growth and all deficiency diseases were particularly severe in the growing age, without considering both of these factors together. Vitamin *E* in particular might yet be shown to have a profound influence on the internal secretions as well as the more obvious functions of the reproductive organs. The increased popularity of vitamin-rich foods, of artificial sun lamps, of transradiant glass windows *et cetera*, their widespread advertising campaigns and the exaggerated statements so typical of modern advertising, put on the health departments a responsibility which had not yet been tackled. If it was the duty of a health department to uphold and test food standards so that the consumer got food of the required quality, then the pure food acts must inevitably have regulations in this direction and health departments must provide the means of biologically testing the claims of various food producers. He could not help feeling that many of the testimonials given by scientific or apparently scientific institutes had been bought and were those rather of an advocate than of a judge. It was to the health departments that they looked for the provision of an impartial referee in this matter.

Dr. Sutton referred to the question of malnutrition in children and said that it was often difficult to get medical practitioners to recognize that errors in diet must be put right. Practitioners were frequently blind to the fact that the child was not receiving the right sort of food. In referring to life in outlying country districts, he said that it was imperative that some provision should be made for people who were compelled to live in those parts. He had repeatedly seen the ill-effects of life in remote districts in male teachers in his department. A young man would go away strong and well and four or five years later he would return a different person. Men appeared to shrink. It was due to deficient diet—few vegetables and little fruit—and health was undermined. He had found that the use of tinned tomatoes was effective in preventing damage to health.

Barcoo rot appeared to be the same as "septic toes" which in the Army in Sinai had reacted so favourably when the patient had been transferred to the sea shore where the Red Cross supplied fresh meat, fruit and vegetables, although previous regular and careful antiseptic treatment at the ambulances had been futile. Investigations by the school oculists in both New South Wales and Victoria had shown during ten years that trachoma had disappeared from the Lower Murray areas from Echuca to Wentworth. The only difference appeared to be the advance of the wheat belt through this area with closer settlement and a more civilized and adequate diet. Dietetic failure might explain the chronic character of the condition.

Dr. Sutton then referred to the subject of constipation in children and said that at the Glenfield Special School they had demonstrated that a properly balanced diet with plenty of roughage combined with a regular habit and plenty of exercise resulted in the elimination of constipation. It was the usual thing in many schools and institutions to consume bottles of aperient medicines in regular use; at Glenfield aperients were seldom, if ever, used. The better class schools, the schools patronized by the members of the medical profession for their own children, were the worst offenders. At these schools the diets given were those that would have obtained two generations before. In conclusion Dr. Sutton said that with the introduction of concentrated preparations of vitamins there was a new problem to be faced. There was such a thing as hypervitaminosis and work should be undertaken in order that the correct dosage of vitamins might be determined.

DR. C. E. CORLETTE said that there was so much to say on the subject that any remarks of his would necessarily be scrappy. For years he had had a grievance against doctors. It was commonly thought that a man was not a real doctor until he found out all the things that a patient liked and then said he must not take them. Many practitioners did this and the result was that the patient lived on a diet with no vitamins and deficient in mineral content. These miserable creatures were held in a cage by the treatment and could never get well. He had told many of these people, victims of medical tradition, to go and have a square meal, that it would give them a big

pain, but that they were to persevere. He had cured a lot of patients in this way. He was quite sure that it was the correct treatment for visceroptosis and possibly for pain simulating gastric ulcer. It should never be forgotten that one of the first results of deficiency of vitamin *B* was loss of all appetite. He thought that by faulty directions to patients doctors were responsible for a great deal of the constipation in the community. They should look for means of supplying what was necessary in the diet without allowing the patient to go to the chemist. He was sure that many of the proprietary food accessories sold to the public were doing harm. Some were frauds. Their ancestors had had a suitable dietary which had gone out of fashion one hundred and fifty years before. He referred to sprouted wheat, the "frumenty" of their ancestors. This was still used in the countries surrounding the Black Sea and food of a similar nature was used in China, namely, sprouted beans. If an effort was made to get wheat to sprout in summer, it would be found that the wheat would get mouldy. The best method was to take a flower pot and to put something into the bottom to prevent the wheat from falling through and then to fill the pot with wheat and to place it under a constant drip of water, such as a dripping tap, after soaking the wheat first of all for twenty-four hours. There would then be no trouble with mould. If they gave this to their children, they would only be reverting to the diet of their ancestors. He had tried the sprouting wheat and beans and could believe that people would soon get used to it. At the same time he confessed that he did not like them much himself. They would certainly act as a cure for constipation. In sprouting plants all the vitamins were abundant, though there was not sufficient mineral content. Certain amino acids, for example tryptophane and lysine, that were absolutely necessary for growth, for health and for life in animals, were poorly supplied in grain and this made grain protein a poor or low-grade protein for food purposes. In such cases the missing amino acids had to be supplied by meat. But the right kind of protein did exist in the germ of grain which, however, was a small and insignificant part of the seed and in breakfast foods, flour, invalid food and so forth the germ was removed in the milling process. The logical and economical way of overcoming the defect of grain protein was to make the germ bigger and to alter the amino acid constitution completely in a favourable direction. This could be done easily by making it grow before use.

Turning to the question of neuritis which was characteristic of beri beri, Dr. Corlette thought that they were too apt to identify deficiency polyneuritis with beri beri as a neuritis. These nervous degenerations occurred in all deficiency diseases. They certainly occurred in pellagra and in rickets, particularly in animals. Bland Sutton had written years before on the question of rickets in monkeys and had described nervous degenerations, "pot belly" and so forth. Dr. Corlette said that he was inclined also to regard pernicious anæmia or some cases of it as a deficiency disease. It certainly had some of the characteristics of deficiency diseases; nervous degenerations were met with in some of the sufferers. It was important to bear in mind the widespread incidence of neuritis and they should not be carried away, when they saw a case of peripheral neuritis, to make a hasty diagnosis of beri beri.

He doubted the wisdom of talking of carbohydrate excess. This was not the only thing which characterized the condition of certain patients. Other things were missing from the diet which ought to be there. There was frequently an insufficiency of mineral salts. It was wrong to fix the attention on one component of the diet and not to consider others.

In regard to pink disease he suggested to those who treated affected children, that they should be given liver. Liver seemed to have a wonderful effect in more than one disease. It cured night blindness and xerophthalmia. He thought that it would cure other things as well. He had tried it in odd ways and had found patients signally different after they had taken it. One patient with vague abdominal pains and discomfort had put on two stone in weight.

As far as rickets was concerned, one of the important things was the nervous degeneration. He found that medical men generally concentrated on the changes occurring at the growing points of the bones. He thought that this was wrong. What did the osteoporosis in rickets mean? He held views about the subject that were somewhat different to those usually put forward. Rickets in his opinion was a derangement of metabolism and the growing points of bone suffered as part of the complaint. Growth was an important vital necessity in the young and Nature tried to protect that function. If there was not enough calcium phosphate to supply the growing bone, signs like those of rickets made their appearance. The long bones became denuded of their calcium and osteoporosis occurred. It had been observed that the rickets might be cured and the osteoporosis get worse. If osteoporosis went on, there would be no rickets. He thought that the hormones or substances of that nature did not always act properly. He thought that there was justification for the view that the breaking down of bone was carried out by the parathyreoid. The parathyreoid principle had been separated and they knew what it did. As a result of increase of parathyreoid secretion the blood calcium rose and one source of this calcium was certainly the bones. He imagined that in health the removal of calcium from the bone was regulated by the parathyreoid. When the parathyreoid was out of action, osteoporosis did not go on and rickets occurred. He thought that vitamin *D* acted on the parathyreoid glands. It was a theory which could be supported by a large amount of evidence.

DR. KENNETH SMITH thanked the readers of the papers and regretted the small attendance. The subject was particularly attractive, as it held out hopes of rescuing certain diseases from the surgeons. He referred to the occurrence of polyneuritis in Lemnos in 1915 and said that it had been a cause of friction between the Number 3 Australian General Hospital and the Army Service Corps who strenuously denied that the ration was at fault. Professor Sir C. J. Martin had been a member of the hospital staff and his opinion had carried much weight. Yeast cakes were obtained through the Lister Institute and the patients were fed on these, Worcestershire sauce being the medium. He also referred to Dr. Harvey Sutton's remarks in regard to trachoma and thought them very important, as the observation was a definite contribution to the study of diet deficiency diseases and was sure to be noted in other countries. Dr. Sutton had mentioned tinned tomatoes as an excellent supplement to the diet of teachers who had to board in small country villages. He had seen reference in the literature to a condition termed "tomato toe" which had arisen in America where large quantities of tinned tomatoes were consumed. He himself had seen two patients in whom the omission of tomatoes from the diet, had resulted in the disappearance of gouty manifestations.

DR. L. R. PARKER said that the practical point was that of all the deficiency diseases the only one with any specific interest to Australia was rickets. Cod liver oil and egg yolk had been mentioned that night as antirhachitics, but he had listened in vain for any reference to butter as a preventive. He asked whether butter did not contain an antirhachitic substance and was informed by Dr. Harvey Sutton that butter from grass-fed cows contained such a substance.

Dr. Parker went on to say that the point was an important one in connexion with infant feeding. At Greycliffe where he had charge of many infants, they used two things as antirhachitics, cod liver oil and butter and found that the children whose feeding was supplemented by butter, did just as well as those to whom cod liver oil was given. This was important when the diet had to be continued at home. Eggs were doubtful in more senses than one and were always expensive. Cod liver oil was also expensive and some children would not take it. It was useless telling a poor mother that she should give her baby the yolks of eggs or cod liver oil. Butter was different; it was cheap, palatable and could be handled easily and safely by parents or guardians of no mental capacity of any description. Most of the babies at Greycliffe were about one to three months old on admission and it was improbable that they had developed rickets before that age and if they did not subsequently

become affected while they were at the institution, it must necessarily be due in the majority to the use of butter. He had failed to detect rickets in any of the babies and he had been supported on one occasion in this observation by Dr. Elsie Dalyell who had examined the children with care. He emphasized his view that butter should be used as an antirhachitic factor in infant feeding. His experience of yeast was limited. He remembered one man who had been associated with yeast manufacture in a large industrial concern and this man was one of the most miserable specimens that he had ever seen.

DR. P. FIASCHI asked whether the high chlorination of the water at Gallipoli had had anything to do with the prevention of scurvy among the troops.

DR. PURDY in reply said that he agreed with what had been said about sprouted wheat and beans. They were in constant use in the Chinese restaurants in Lower Campbell Street, Sydney. He also referred to an experience during the South African War. An old doctor at Capetown had told him that veldt sores could be cured if the patient ate one pound of grapes a day and he had found that this was so. In Sinai in 1906 he had seen ulcers similar to veldt sores and barcoo rot and he had been given to understand that a tin of pineapple juice was a specific. In regard to butter he could not agree that it was cheap. The people of Sydney paid threepence a pound more than the people in London paid for the same butter. He thought it was a pity that it could not be sold in Sydney at a more reasonable figure. As far as the *Pure Food Act* and regulations were concerned, he thought that it should be emphasized that a guarantee given by a manufacturer under this act had to do only with standards and preservatives, the guarantee was not based on a biological or physiological test. He thought that there was room for much more investigation, especially in regard to infant and invalid foods.

DR. WILLCOCKS in reply thanked the other speakers for their remarks. The subject was so enormous that he had cut out all but essentials. He had said nothing about the prevention of rickets. He thought that most children had a diet which was deficient in milk. He agreed that butter was a good substitute. He had not been aware that infection played so important a part as Dr. Sutton had said. In regard to Dr. Corlette's remarks about carbohydrate excess and deficiency he wished it to be understood that he (Dr. Willcocks) had spoken of a diet consisting of carbohydrates only.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Collins, Kevin James, M.B., Ch.M., 1926 (Univ. Sydney), Glenrock Lodge, Edgecliff.

Sender, Isidor Harry, M.B., B.S., 1928 (Univ. Sydney), Sydney Hospital.

Gleeson, William Sydney, M.B., Ch.M., 1926 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

Baxter, Ernest Joel, M.B., Ch.M., 1925 (Univ. Sydney), 12, Tranmere Street, Drummoyne.

Correspondence.

SUBOCCIPITAL DERMOID CYSTS.

SIR: May I be allowed to comment on the report of a discussion of the Melbourne Pædiatric Society on a case of suboccipital dermoid cyst, published in your issue of October 6, 1928 (page 447). While it is true that condensed reports of meetings of clinical societies may often misrepresent the speakers' remarks, yet in this particular report so many inaccurate statements are placed before your readers that some criticism is necessary. From perusal of your report the general practitioner would be entitled to assume that at present the prognosis of subtentorial tumours in the young is almost hopeless. This is far from true.

Dr. C. H. Osborn states that in Cushing's series of intracranial tumours in children the prolongation of life had been only six months after operation. It is evident from his other remarks that Dr. Osborn refers to an article on "The Intracranial Tumours of Preadolescence" (*American Journal of Diseases of Children*, 1927, Volume XXXIII, page 551) in which Cushing reports his experiences with 154 verified intracranial tumours in children under fifteen years of age. In this series the prolongation of life after operation for the various forms of glioma has been carefully estimated. For the astrocytomas, the most common and benign variety of glioma, the survival period is six years or more, in spite of several operative fatalities in the earlier cases of the series. Some cases, indeed, are alive twenty years after operation. It is only in reference to the medulloblastomas, the most malignant of the gliomas, that Cushing states: "We have estimated (probably too conservatively) that the patients . . . do well, on the average, for six months after operation." The clause in parenthesis has proved to be justified by more recent observations of the beneficial effects of radio-therapy in this variety of glioma. For tumours other than gliomas the prognosis after operation is even better than for the astrocytomas, since most of them are benign tumours.

Dr. Osborn is reported to have said that it is too late to operate after hydrocephalus has developed, but surely this must be a misrepresentation of his words, for in most subtentorial tumours the clinical picture is one of hydrocephalus from the beginning of the illness. Indeed, subtentorial tumours in children are notorious for the paucity of their localizing signs and it is not unusual to explore the cerebellum of a child showing slight enlargement of the head, papillœdema and other signs of hydrocephalus when the only localizing signs present are those of slight hypotonia and incoordination. In such cases the tumour is usually found in the vermis. It is never "too late to operate" in these cases and success has even been known to follow operations begun under artificial respiration. What is of first importance is that the operation should be undertaken before vision is seriously damaged.

Dr. H. D. Stephens's experience that removal of subtentorial tumour is almost impossible is in agreement with the experience of most other neurological surgeons until recent years, but it is not an accurate statement of the present position. In Dr. Cushing's clinic, for example, during the year 1926-1927 when it was my privilege to act as assistant resident surgeon to the clinic, subtotal or complete removal of subtentorial tumours in children was accomplished in almost every case and with a very low death rate. Lack of space prevents me from recounting the methods of operation, but I have described these fully in a report to the Medical Research Council, now in the press. It is scarcely necessary to mention that even when it is not possible or advisable to remove the tumour, a liberal suboccipital decompression, carried out with due care and hæmostasis, gives strikingly beneficial results, particularly in cases of benign tumour, among which may be included dermoid cyst.

Dr. Stephens's assertion that tuberculoma is the commonest tumour on the other side of the world agrees with that of Allen Starr who towards the end of last century collected cases from the literature and found that tuberculomas comprised one-half of all intracranial tumours. Starr's estimates have largely influenced the figures given by subsequent writers, but they are not confirmed by the most recent investigations. Among one thousand cases of verified intracranial tumours at Cushing's clinic Van Wagenen found only fourteen tuberculomas. Neurosurgical material, however, is to a certain extent selected and I have therefore collected the verified intracranial tumours in the records of the London Hospital for the years 1916 to 1921. Among one hundred "tumours" (that is, neoplasms, parasitic cysts and granulomas) there are only thirteen tuberculomas. This figure probably provides a fairly accurate estimate of the fre-

quency of tuberculoma in the population of London and there is reason to hope, therefore, that tuberculoma of the brain is becoming less common and that in this as in other respects the prospect for cases of intracranial tumours is not so gloomy as the discussion of the Melbourne Pædiatric Society implied.

Yours, etc.,

HUGH CAIRNS.

London Hospital,
Whitechapel, E.1,
November 23, 1928.

OBSTETRICAL PRACTICE.

SIR: In your issue of January 5 there is published an article by Dr. Leighton Kesteven entitled "Prophylactic Obstetrical Practice." It is difficult to be certain to what the prophylaxis refers and I would suggest an alternative title: "The Renaissance of *Accouchement Forcé*."

Dr. Kesteven, to emphasize his contentions, requires the aid of discourtesy and personal abuse in his reference to a very sincere effort to add something of assistance to a world-wide problem and so he has answered "much more solemn nonsense" by much more "ludicrous rubbish." Perhaps one might be entitled to regard it as an instance of the cap fitting most excellently after having been worn for more than ten years. The almost routine manual or forcible dilatation of the cervix followed by forceps delivery is nothing less than *accouchement forcé*, an obstetric operation which is obsolete, without scientific foundation and universally condemned and universally associated with high maternal and fœtal mortality.

Dr. Kesteven's management of the placental stage reveals an ignorance of the mechanism and physiology of this stage. Moreover, by his utter disregard and apparent ignorance of the principles of the science and the art of obstetrics this noble science has once again been dragged through the mud, even though subsequently cleansed by that odorous panacea lysol.

Dr. Kesteven is at variance with such authorities as Joseph De Lee, Whitridge Williams, B. P. Watson, Hastings Tweedy, Gibbon FitzGibbon and it might interest him to know that his quotation from my article was in substance contained in the Report of the Committee of Maternal Welfare of the American Association of Gynæcologists, Obstetricians and Abdominal Surgeons.

Dr. Kesteven is also apparently ignorant of the technique of the various world famed maternity centres and of the conclusive analysis of cases published in their annual reports. These conclusively show that all operative procedures including forceps delivery are followed by a higher morbidity rate than is natural delivery. This has also been conclusively demonstrated at the Women's Hospital, Melbourne, where in the last three years about ten thousand deliveries have taken place. Here the honorary obstetric staff have each year expressed their satisfaction at the very low proportion of forceps deliveries. I should also like to point out that Dr. Kesteven's figures cannot be sufficiently well analysed as the proportion of *primiparæ* to *multiparæ* and the average duration of labour in each class, *inter alia*, have not been recorded.

The quotations denouncing meddlesome midwifery are those of eminent obstetricians and it must be much more wearisome for them to announce them than for those who fail to heed and who ignore them. To one who has travelled the world learning the considered views and methods of the best authorities and who has been accepted as a teacher in obstetrics, the radical views held by Dr. Kesteven are intolerable and I am firmly of the opinion that the overwhelming majority of medical practitioners practise conservative and scientific midwifery to the best of their ability and allowing for variation of conditions.

The failure in the reduction of the maternal and fœtal mortality rates is accounted for in this article; a few others are showing the same utter disregard for well founded scientific principles and unhappily their results are not so fortunate as are Dr. Kesteven's.

The statement of Dr. Wawn referred to is quite reconcilable with our accepted views, *videlicet* that a necessary condition for the application of forceps is that labour has advanced to the second stage.

Sydney Morris does refer to the improvement in technique, but in almost the same breath he refers to the necessity for the limitation of the various operative procedures.

Here again technique, asepsis and antisepsis have been thoroughly thought out, investigated and applied at all the important maternity hospitals and are as perfection when compared to the crude routine of Dr. Kesteven.

I have been unable to find any records of Dr. Kesteven's research on the vaginal flora and his emphatic opinion that no pathogenic organisms are normally present is on the same plane of accuracy and logic as the rest of his paper.

In conclusion it is with a feeling of intense satisfaction that I have been charged as a champion of conservative obstetrics by Dr. Kesteven who has associated my humble self alongside those others in whose path I have endeavoured to follow.

Yours, etc.,

HUBERT JACOBS.

M.D., B.S. (Melb.), F.R.C.S.Ed., D.G.O. (Dublin), L.M. Rotunda Hospital, F.C.S.A.; late Medical Superintendent, Women's Hospital Melbourne; late Honorary Obstetric Surgeon, Women's Hospital; Honorary Gynæcologist to Out-Patients, St. Vincent's Hospital, Melbourne.

January 8, 1929.

CLASSIFICATION OF DISEASES.

SIR: Many important points are made in the informative address by Sir George Knibbs on "The International Classification of Disease and Causes of Death" that was published in your columns on January 5. There appears, however, some need to correct an impression left by Sir George Knibbs's statement that: "It is twenty-one years since attention was called to the need for insuring correct certification of death. As far as I am aware nothing has been attempted."

As a matter of fact there has been a steady advance within recent years Continued effort has been made to insure on the one hand improved certification by the medical attendant and on the other hand comparable and complete compilation and tabulation of mortality statistics. The question has been fully discussed and coordinated methods adopted through the periodical conferences of Commonwealth and State statisticians. A further important step was the joint conference of statisticians with the Federal Health Council in March, 1928. Amongst the more important resolutions adopted that related to the certification and classification of causes of death were the following:

Certificate of the Cause of Death.

(*a*) That for the purpose of classification of causes of death and for other purposes it is desirable that a medical officer be associated with the staff of the Commonwealth Statistician in the compilation of vital statistics.

(*b*) It is considered desirable that the practice of referring to the certifying doctors medical certificates containing indefinite or misleading terms should be adopted in all States and carried out as extensively as is necessary.

(*c*) That the Director-General of Health and the Statistician of the Commonwealth prepare a draft leaflet for insertion in each volume of blank medical certificates of death conveying information to the medical practitioner of points to be observed in connexion with the certification, and this Conference recommends the use of such draft leaflet by the Registrar-General of each State.

Confidential Notification of the Cause of Death.

(*a*) That consideration should be given by the Australian registration authorities to the system of confidential certification of cause of death employed for some years in Switzerland and recently adopted in Holland.

(*b*) That the Director-General of Health and the Commonwealth Statistician be appointed a subcommittee to discuss the matter with the several registration authorities in Australia and with the Federal Committee of the British Medical Association in Australia.

Verification of Cause of Death.

A suggestion by Dr. Ramsay Smith that the fact of the verification of the cause of death after *post mortem* examinations should be recorded was referred to the subcommittee which is to deal with the leaflet to accompany books of certificates of death.

Other resolutions related to suggested variations in the list of causes of death and the tables that should be compiled for publication.

There is a popular conception that the resolutions adopted by official conferences are for the most part pious expressions of opinion that are usually barren of results. It is therefore definitely gratifying to record that some practical result is following every item of the resolutions adopted by this conference. As noted by Mr. Wickens in reply to the discussion that followed Sir George Knibbs's address (see page 29) "it has been decided that the adoption of the English system would be a step in the right direction, especially the inclusion of detailed directions in the death certificate book." This English system, initiated under the *Registration Act* of 1926 marks a very definite advance, embodying many of the advantages of the so-called confidential system. An admirable summary of this system appeared in *The British Medical Journal* for June 18, page 1118. Consideration is now being given to the practical details involved in the adoption of such a uniform system for Australia.

Close cooperation between the Commonwealth Bureau of Census and Statistics and the Commonwealth Department of Health provides for mutual consultation. This cooperation has extended to frequent communication with the committees that are engaged in the preliminary work preparatory to the forthcoming revision of the International List of Causes of Death—the Commission of Expert Statisticians of the Health Section of the League of Nations and the *Commission Mixte de Statistique Sanitaire* of the International Statistical Institute. It is hoped that Australia will be suitably represented at the International Conference that will assemble at Paris in October next to discuss the revision.

While this advance is being maintained and an improved and uniform system formulated for Australia, emphasis should still be laid upon Sir George Knibbs's closing words to his audience of medical men: "I now leave the matter in your hands." Since the whole value of any system of mortality statistics rests ultimately on correct certification, the success or failure of any new system must depend on the degree of cooperation forthcoming from the practising profession.

Yours, etc.,

J. H. L. CUMPSTON, M.D.,
Director-General of Health.

Canberra.
January 9, 1929.

POST-GRADUATE FACILITIES.

SIR: I wish to inquire through the columns of your paper if there are any facilities in city hospitals for country doctors to obtain tuition in special branches of medical science.

I know that the general hospitals are always open to visiting doctors who may watch proceedings therein, and I know also of the annual post-graduate courses.

However, if a doctor desires to specialize, he must have efficient tuition and a practical training in his speciality. Surely a few years in a general practice is the best grounding for the future specialist. Yet most men after a few years in general practice have acquired domestic responsibilities which make a trip to Europe financially out of the question, but they could spend several months in an Australian city to obtain a grounding in their speciality.

At present it seems that the only way to become a specialist in the future is to settle in the city, so as to belong to the staff of a city hospital.

Doctors with special knowledge who could work together in teams, are badly needed in many of our country towns, but how can these men obtain their training? Some have enough native genius to specialize with the aid of books, but such men are few indeed.

Could it not be arranged that a certain number of clinical assistantships be made available for country doctors? Could not special courses be arranged for tuition in such subjects as cystoscopy, the administration of intratracheal ether and the use of diathermy?

Yours, etc.,

"COUNTRY PRACTITIONER."

January 17, 1929.

Post-Graduate Work.

POST-GRADUATE WORK IN BERLIN.

WE have been asked to make the following announcements in regard to the post-graduate courses that have been arranged by Kaiserin Friedrich-Stiftung in conjunction with the Faculty of Medicine of the University of Berlin and the committee of lecturers. The courses are partly permanent and partly held during the months of March and April, 1929.

Permanent Courses.

(i) Regular post-graduate courses lasting two to four weeks;

(ii) Courses given to those acting as assistants in clinics, hospitals and laboratories, lasting for two to three months. These courses are suitable for practitioners desirous of carrying out practical work under systematic supervision.

Courses in March and April.

March 4 to 16, 1929.—Course in general medicine, especially in cardiology. Professor von Bergmann, Professor Dresel, Professor F. Klemperer, Professor Munk, Professor Rosin, Professor V. Schilling, Professor Schlayer and Professor Straus. Fee: 80 reichsmarks.

March 18 to 23, 1929.—Special course in Urology. Professor Bärtzner, Professor Casper, Professor E. Joseph, Professor von Lichtenberg, Professor Ringlieb, Professor Rumpel. Fee: 75 reichsmarks.

March 18 to April 13, 1929.—Special courses on Breast and Stomach Surgery. Professor Bärtzner, Professor Bier, Professor Borchardt, Professor Katzenstein, Professor Kisch, Professor Mühsam, Professor Sauerbruch, Professor Unger. Fee: 75 reichsmarks.

April 14 to 21, 1929.—Special course in Röntgenology in surgical diseases. Professor Chaoul, Professor Max Cohn, Professor Cramer, Professor Frik, Professor Hintze, Professor Lazarus, Professor Levy-Dorn, Professor Munk and Professor Rumpel. Fee: 100 reichsmarks.

March and April, 1929.—Single courses on all branches of medicine and surgery, including practical work.

The courses are held in German, but many of the professors are able to lecture in English, French and Spanish.

Full particulars concerning suitable lodging, the approximate cost of living during the period of the various courses and the like can be obtained from the information bureau of the Kaiserin Friedrich-Haus für das ärztliche Fortbildungswesen, Berlin, N.W. 6 Luisenplatz 2 to 4.

Obituary.

PERCIVAL JAMES CAMPBELL.

IN our issue of December 8, 1928, we recorded the death of Percival James Campbell at Trafalgar, Victoria.

Percival James Campbell was born at Tallangatta, Victoria, on May 26, 1894. He was the son of J. G. Campbell, of Clewes Street, South Yarra, formerly a teacher in the Education Department. Percival James Campbell received his early education in East Malvern, where he won a Government scholarship at the end of 1907. In 1908 he entered Scotch College and at this school he secured a further scholarship in 1911, entitling him to five years' tuition at the University of Melbourne. He commenced his medical studies in 1912 and in 1916 he graduated in medicine and surgery. Immediately after graduation he enlisted in the Australian Imperial Forces for active service and went overseas. He served as Medical Officer to the Fifth Brigade of the Field Artillery. After the armistice he spent some time at Ancoats Hospital, Manchester, carrying out post-graduate work. He then returned to Victoria and practised at Castlemaine, Natimuk and finally at Trafalgar. His tastes were literary and artistic rather than athletic; moreover, war service had so affected his health that he was unable to take violent exercise. He suffered at various times from a heart affection which appears to have been progressive and to have led to his untimely death.

During his university career Percival James Campbell contributed literary articles and black and white sketches to various papers. He held the position of editor of *The Speculum* in 1915-1916 and also to *The Journal of the Medical Students' Society*. He also assisted in illustrating the second edition of Dr. George Horne's text book on practical gynæcology. This work was not completed when he enlisted for active service. He married a daughter of the late J. Beckinsale, of Castlemaine, who survives him. He was a freemason and took an active part in municipal and social affairs. He was a successful practitioner, a kind friend and a man of high ideals. He was loved and respected by his friends and all his patients.

Our deepest sympathy is extended to his widow, his parents and his brother, Dr. J. C. Campbell, of Sale.

Dr. Frank Stone writes:

"The news of Dr. P. J. Campbell's death came as a shock to his friends and colleagues. I had known him since a very happy association on the resident staff of Ancoats Hospital, Manchester, where he endeared himself to all. His kindly humour and generous nature made him deservedly popular with patients and colleagues alike and we now feel that we have lost a very good friend."

Naval and Military.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazettes*, Numbers 112, 131, 135 and 140, of October 11, November 29, December 6 and 20, 1928.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointments.—Donald Dunbar Coutts, M.B., B.S., D.S.O., is appointed Surgeon Lieutenant, dated 15th October, 1928.

Appointment.—Clive Henry Reynolds James, M.B., B.S., is appointed Surgeon Lieutenant, with seniority in rank of 1st September, 1927, and as Sub-District Naval Medical Officer at the Port of Geelong. Dated 1st December, 1928.

Promotion on Retired List.—Surgeon Lieutenant William Keverall McIntyre is promoted to the rank of Surgeon Lieutenant-Commander, with seniority in rank of 2nd December, 1927.

Termination of Appointments.—The appointments of the following officers are terminated:—Surgeon Lieutenant-Commanders Francis Henry Vivian Voss, James Henry Sleeman; Surgeon Lieutenants George William Mason, Geoffrey Hampden Vernon.

Termination of Appointment.—The appointment of Surgeon Lieutenant-Commander (Retired List) Fossey James Newman as Sub-District Naval Medical Officer at the Port of Geelong is terminated. Dated 30th November, 1928.

Royal Australian Naval Volunteer Reserve.

Termination of Appointment.—The appointment of Surgeon Lieutenant Clive Henry Reynolds James is terminated. Dated 30th November, 1928.—(Ex. Min. 225.)

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps.

Lieutenant (provisionally) C. B. Boddington is transferred to the Australian Army Medical Corps Reserve and to be Honorary Lieutenant, 7th September, 1928.

The provisional appointment of Captain H. S. Roberts is terminated, 20th August, 1928. Honorary Captain H. S. Roberts is re-appointed from the Australian Army Medical Corps Reserve, and to be Captain (provisionally), 21st August, 1928. *To be Captains (provisionally).*—Ambrose John Foote and Wilfred Park, 20th August, 1928, and 19th September, 1928, respectively.

To be Captain (provisionally) supernumerary to the establishment pending absorption—John Lloyd Simmonds, 26th November, 1928.

Australian Army Medical Corps Reserve.

The notification of the retirement of Captain R. H. Leeds, which appeared in Executive Minute No. 150/1928, promulgated in *Commonwealth Gazette*, No. 73, of 26th July, 1928, is cancelled.

Second Military District.

Australian Army Medical Corps.

Captain L. T. Allsop, M.C., is appointed from the Australian Army Medical Corps Reserve, 27th August, 1928. Major W. T. Newton is transferred to the Australian Army Medical Corps Reserve, 14th September, 1928.

To be Captain (provisionally) supernumerary to the establishment pending absorption.—Roland Edward Longworth, 13th September, 1928. *To be Captain (provisionally).*—John Richard Phillips, 13th September, 1928. Captain (provisionally) G. Cummins is brought on the authorized establishment, 1st September, 1928.

Major T. M. Furber is appointed to command the 9th Field Ambulance, 1st September, 1928. Major W. Vickers, D.S.O., is appointed to command the 2nd Field Hygiene Section, 14th September, 1928. The provisional appointment of Captain J. C. Belisario is confirmed.

To be Lieutenant-Colonel.—Major W. R. C. Beeston, 1st September, 1928. Lieutenant-Colonel P. Fiaschi, O.B.E., V.D., from the command of the 9th Field Ambulance, is appointed to command the 4th Cavalry Field Ambulance, 1st September, 1928.

Captains (provisionally) A. E. M. Moir and S. C. M. Hiatt are brought on the authorized establishment, 1st November, 1928. The provisional appointment of Captain R. B. Austin is terminated, 3rd October, 1928.

To be Captain (provisionally).—Robert Blackie Austin, 4th October, 1928. Captain N. H. Meacle is transferred to the Australian Army Medical Corps Reserve, 15th September, 1928.

Australian Army Medical Corps Reserve.

Honorary Captain A. C. McArthur is retired, 20th September, 1928.

Honorary Captains H. L. Jones and R. M. Kinross are retired, 30th November, 1928, and 4th December, 1928, respectively.

Lieutenant V. R. Bellemey is retired, 1st December, 1928.

Third Military District.

Australian Army Medical Corps.

Captain J. Morlet is appointed from the Australian Army Medical Corps Reserve, 10th September, 1928; the provisional appointment of Captain E. A. Daley is confirmed; Lieutenant-Colonel J. K. Adey, O.B.E., is transferred to the Unattached List, 1st August, 1928.

To be Lieutenant (provisionally) supernumerary to the establishment, pending absorption.—Glen Albyn Martin Knight, 10th September, 1928; Captain J. I. Connor is transferred to the Australian Army Medical Corps Reserve, 1st September, 1928.

To be Lieutenants (provisionally).—Lawrence Edward Odlum, Alexander George Mancy, and Norman Josiah Solomon, 26th September, 1928; and Egbert Armytage Cuninghame Farran, 4th October, 1928. Lieutenant (provisionally) N. J. Solomon is supernumerary to the establishment pending absorption, 26th September, 1928. The commission of Captain E. A. Daley is terminated on his appointment to the Royal Australian Air Force, 15th July, 1928.

To be Major.—Captain K. C. Purnell, M.C., 5th October, 1928.

To be Major.—Captain D. L. Yoffa, 11th October, 1928; Lieutenant-Colonels H. J. Williams, D.S.O., and E. W. Gutteridge are brought on the authorized establishment of Lieutenant-Colonels, 1st November, 1928; Lieutenant-Colonel F. E. Keane, M.C., is supernumerary to the establishment of Lieutenant-Colonels with pay and allowances of Major, 16th July, 1928.

Majors J. A. O'Brien, G. S. Robinson, M.C., and D. C. Pigdon are appointed from the Australian Army Medical Corps Reserve, 1st August, 1928.

Captain R. W. Lawrence is appointed from the Australian Army Medical Corps Reserve, 26th November, 1928.

Australian Army Medical Corps Reserve.

To be Honorary Major.—Frank Clare Wilkinson, 1st October, 1928.

Honorary Major W. E. Davies is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 1st October, 1928.

Captain W. F. Orr is placed upon the Retired List with the honorary rank of Major and with permission to wear the prescribed uniform, 15th November, 1928; the resignations of Captain A. E. Brown, Honorary Captains C. G. McAdam and A. S. Robertson and Honorary Lieutenant W. A. E. Graham of their commissions are accepted, 15th November, 1928; Honorary Captain T. A. Wilson is retired, 10th November, 1928.

Award of the Colonial Auxiliary Forces Officers' Decoration.

Australian Army Medical Corps.—Lieutenant-Colonel J. J. Black, D.S.O.

Australian Army Medical Corps.—Lieutenant-Colonel N. L. Speirs.

Unattached List.

Lieutenant-Colonel W. W. W. Chaplin is transferred to the Australian Army Medical Corps Reserve, 5th October, 1928.

Fourth Military District.

Australian Army Medical Corps.

The provisional appointment of Captain J. E. Porter, M.M., is confirmed.

To be Captain (provisionally).—Eric Frank Gartrell, 7th November, 1928.

The provisional appointment of Captain F. R. Hone is terminated, 25th October, 1928. *To be Captain (provisionally).*—Frank Raymond Hone, 26th October, 1928.

Australian Army Medical Corps Reserve.

Captain J. S. Proctor is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 23rd September, 1928.

Honorary Captain J. B. Gunson is retired, 18th December, 1928.

Award of the Colonial Auxiliary Forces Officers' Decoration.

Unattached List.—Lieutenant-Colonel E. A. H. Russell.

Fifth Military District.

Australian Army Medical Corps.

To be Major.—Captain J. R. Donaldson, 31st August, 1928.

Major L. A. Hayward is transferred to the Australian Army Medical Corps Reserve, 10th November, 1928. The provisional appointment of Captain H. L. Johnston is confirmed.

Honorary Captain J. L. Day is appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally), 14th November, 1928; the provisional appointment of Captain L. G. Male is confirmed.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Force: Medical Branch.

Resignation.—The resignation of Wing Commander A. P. Lawrence, M.C., is accepted, 24th September, 1928.

Citizen Force: Medical Branch.

Appointment.—To Wing Commander with seniority as from 1st July, 1927, Arthur Poole Lawrence, M.C., and to receive pay at the rate of £600 per annum whilst employed as Deputy Director Medical Services (Air), 25th September, 1928.—(Ex. Min. No. 201.)

THE WILLIAM GIBSON RESEARCH SCHOLARSHIP FOR MEDICAL WOMEN.

MISS MAUD MARGARET GIBSON has placed in the hands of the Royal Society of Medicine a sum of money sufficient to provide a scholarship of the yearly value of £292, in memory of her father, the late Mr. William Gibson, of Melbourne, Australia. The scholarship is awarded from time to time by the Society to qualified medical women who are subjects of the British Empire, and is tenable for a period of two years, but may in special circumstances be extended to a third year. The next award will be made in June, 1929.

In choosing a scholar, the Society will be guided in its choice "either by research work already done by her or by research work which she contemplates. The scholar shall be free to travel at her own will for the purpose of the research she has undertaken."

There is no competitive examination nor need a thesis or other work for publication or otherwise be submitted. The Society has power at any time to terminate the grant if it has reason to be dissatisfied with the work or conduct of the scholar.

Applications should be accompanied by a statement of professional training, degrees or diplomas and of appointments, together with a schedule of the proposed research. Applications must be accompanied by testimonials, one as to academical or professional status, and one as to general character. Envelopes containing applications should be marked on top left-hand corner: "William Gibson Research Scholarship" and should be addressed to Mr. G. R. Edwards, Secretary, Royal Society of Medicine, 1, Wimpole Street, London, W.1, and be received not later than Saturday, June 1, 1929.

THE ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH.

THE annual meeting of the Royal College of Physicians of Edinburgh was held on December 6, 1928. Dr. Robert A. Fleming was reelected President for the ensuing year;

Dr. Robert Thin was nominated Vice-President; Dr. G. Lovell Gulland, Dr. John Orr, Dr. William Fordyce, Dr. Edwin Bramwell and Dr. A. Fergus Hewat were elected to form the Council.

At an extraordinary meeting held on the same day Dr. William Russell and Dr. John Orr were reelected representatives of the College on the Board of Management of the Royal Infirmary of Edinburgh.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Buchanan, Angus Dugald, M.B., Ch.B., 1911 (Univ. Glasgow), Yeppoon.

Simmonds, John Lloyd, M.B., B.S., 1928 (Univ. Melbourne), Brisbane.

Skinner, Harrison Booth, M.B., B.S., 1928 (Univ. Melbourne), Brisbane.

Spencer, John Clark, M.B., B.S., 1925, M.D., 1928 (Univ. Melbourne), Commonwealth Health Department, Rockhampton.

Williams, Noel Swifte, M.B., B.S., 1928 (Univ. Melbourne), Brisbane.

Townsend, Alan Robert, M.B., B.S., 1923 (Univ. Melbourne), Ipswich.

Restorations to the Register:

Dodson, George Hirst, M.B., Ch.M., 1921 (Univ. Sydney), Roma.

Luddy, John Joseph, M.B., 1909, Ch.M., 1912 (Univ. Sydney), Brisbane.

VICTORIA.

THE undermentioned have been registered under the provisions of Part I of the *Medical Act*, 1915, of Victoria, as duly qualified medical practitioners:

Woodruff, Harold Addison, M.R.C.S. (England), L.R.C.P. (London), 1921, The University of Melbourne.

Amies, Arthur Barton Pilgrim, L.R.C.P. *et* S. (Edinburgh), L.R.F.P.S. (Glasgow), 1928, 32, Collins Street, Melbourne.

Burns, Hugh Matheson, M.B., B.S., 1926 (Univ. Melbourne), 714, Inkerman Road, Caulfield.

Cole, John Bazil, L.R.C.P. *et* S. (Edinburgh), L.R.F.P.S. (Glasgow), 1928, 3, McHenry Street, East St. Kilda.

Farran-Ridge, Clive, M.B., 1915, Ch.M., 1917 (Univ. Sydney), Dip.P.M. (London), 1921, Commonwealth Bank, Collins Street, Melbourne.

Rowe, Cecil Hartley, L.R.C.P. *et* S. (Edinburgh), L.R.F.P.S. (Glasgow), 1928, St. George's Road, Toorak.

Searls, John Robert, M.B., B.S., 1928 (Univ. Melbourne), 28, Royal Avenue, Glenhuntly.

Staley, Dorothy, M.B., Ch.B., 1923 (Bristol), Natya, *viâ* Swan Hill.

Stewart, Charles Edward, M.B., Ch.B., 1928 (Glasgow), c.o. Armytage, Cheritta, Branxholme.

Additional deplima registered:

Cooper, Eric Leonard, M.D., 1927 (Univ. Melbourne).

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Boddington, Charles Blair, M.B., Ch.M., 1925 (Univ. Sydney), Clayfield, Brisbane.

Leslie, James Stuart, M.R.C.S. (England) 1915, L.R.C.P. (London) 1915, Cremorne.

Livingstone, William Gordon, M.B., B.S., 1924 (Univ. Melbourne), Murwillumbah.

Odlum, Lawrence Edward, M.B., B.S., 1928 (Univ. Melbourne), c.o. A.M.P. Society, Pitt Street, Sydney.

Books Received.

A HANDBOOK ON VENEREAL DISEASES FOR NURSES AND OTHERS ENGAGED IN THE ROUTINE TREATMENT OF THESE DISEASES, by W. Turner Warwick, F.R.C.S.; 1928. London: Faber and Gwyer, Limited. Crown 8vo., pp. 232 with illustrations. Price: 6s. net.

TAKING THE DOCTOR'S PULSE AND ANOTHER ESSAY, by J. F. Montague, M.D., F.A.C.S.; 1928. Philadelphia: J. B. Lippincott Company. Crown 8vo., pp. 103. Price: $1.00 net.

CHEMICAL REACTIONS AND THEIR EQUATIONS: A GUIDE FOR STUDENTS OF CHEMISTRY, by Ingo W. D. Hackh; Second Edition, Revised; 1928. Philadelphia: P. Blakiston's Son and Company. Crown 8vo., pp. 145.

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ROENTGENOLOGY: ITS EARLY HISTORY, SOME BASIC PHYSICAL PRINCIPLES AND THE PROTECTIVE MEASURES, by G. W. C. Kaye, O.B.E., M.A., D.Sc., F.Inst.P.; 1929. New York: Paul B. Hoeber, Incorporated. Crown 8vo., pp. 171, with illustrations. Price: $2.00 net.

AN INDEX OF SYMPTOMATOLOGY, by Various Writers; Edited by H. Letheby Tidy, M.A., M.D. (Oxon.), F.R.C.P. (London); 1928. Bristol: John Wright and Sons, Limited. Royal 8vo., pp. 722, with illustrations. Price: £2 2s. net.

A TEXTBOOK OF MEDICINE, by Various Authors; Edited by J. J. Conybeare, M.C., M.D. (Oxon.), F.R.C.P.; 1929. Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 990, with illustrations. Price: 22s. 6d. net.

CHRONIC (NON-TUBERCULOUS) ARTHRITIS: PATHOLOGY AND PRINCIPLES OF MODERN TREATMENT, by A. G. Timbrell Fisher, M.C., F.R.C.S. (England); 1929. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 245, with illustrations. Price: 25s. net.

ACUTE INFECTIOUS DISEASES, by Jay Frank Schamberg, A.B., M.D., and John A. Kolmer, M.Sc., M.D., Dr.P.H., D.Sc., LL.D.; Second Edition, Thoroughly Revised; 1928. Philadelphia: Lea and Febiger. Royal 8vo., pp. 900, with illustrations. Price: $10.00 net.

SERUM DIAGNOSIS BY COMPLEMENT-FIXATION WITH SPECIAL REFERENCE TO SYPHILIS: THE PRINCIPLES, TECHNIQUE AND CLINICAL APPLICATIONS, by John A. Kolmer, M.S., M.D., Dr.P.H., D.Sc., LL.D.; 1928. Philadelphia: Lea and Febiger. Royal 8vo., pp. 602, with illustrations. Price: $7.00 net.

PARTNERSHIPS, COMBINATIONS AND ANTAGONISMS IN DISEASE, by Edward C. B. Ibotson, M.D. (London), B.S.; 1929. Philadelphia: F. A. Davis, Company. Demy 8vo., pp. 348, with illustrations. Price: $3.50 net.

THE ROENTGEN DIAGNOSIS OF ECHINOCOCCUS TUMORS: A STUDY, by Gunnlaugur Claessen; 1928. Stockholm: P. A. Norstedt and Söner. Crown 4to., pp. 155, with illustrations.

THE TREATMENT OF VARICOSE VEINS BY INTRAVENOUS INJECTIONS, by J. D. P. McLatchie, M.D., C.M.; 1928. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 58. Price: 3s. 6d. net.

TUBERCULIN IN PRACTICE: ITS VALUE IN THE TREATMENT OF EARLY TUBERCULOSIS AND ASTHMA, by F. E. Gunter, D.S.O., M.D. (Edinburgh), Lieut.-Colonel R.A.M.C. (Retired); 1929. London: The Gregg Publishing Company, Limited. Demy 8vo., pp. 112. Price: 7s. 6d. net.

DIABETIC SURGERY, by Leland S. McKittrick, M.D., F.A.C.S., and Howard F. Root, M.D.; 1928. Philadelphia: Lea and Febiger. Royal 8vo., pp. 269, with illustrations. Price: $4.25 net.

Diary for the Month.

JAN. 25.—Queensland Branch, B.M.A.: Council.
JAN. 29.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Professor H. H. Woollard (B.M.A.) has been appointed Honorary Consulting Anatomist at the Adelaide Hospital, South Australia.

* * *

Dr. J. R. S. G. Beard (B.M.A.) has been appointed Honorary Gynæcologist at the Adelaide Hospital, South Australia.

* * *

Dr. Henry John Taylor (B.M.A.) has been appointed a Member of the Queensland Medical Board.

* * *

Dr. Charles Mitford Lilley (B.M.A.) and Dr. Henry Byam Ellerton have been appointed Members of the Nurses' and Masseurs' Registration Board of Queensland.

* * *

Dr. C. H. Wesley (B.M.A.) has been appointed Honorary Assistant Surgeon at the Royal Alexandra Hospital for Children, Sydney.

* * *

Dr. D. G. R. Vickery (B.M.A.) has been appointed Honorary Relieving Assistant Surgeon at the Royal Alexandra Hospital for Children, Sydney.

* * *

Dr. Charleton Yeatman has been appointed Acting Medical Officer to His Majesty's Gaol at Mount Gambier, South Australia.

* * *

Dr. Eugene McLaughlin (B.M.A.) has been appointed Deputy Director of the Laboratory of Bacteriology and Pathology at the Adelaide Hospital, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

ECHUCA DISTRICT HOSPITAL: Resident Medical Officer.

MACKAY HOSPITALS BOARD: Resident Medical Officer.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Honorary Assistant Radiographer, Assistant Medical Superintendent.

ROYAL SOCIETY OF MEDICINE, LONDON, ENGLAND: William Gibson Research Scholarship.

TERRITORY OF PAPUA: Government Medical Officer.

THE ADELAIDE CHILDREN'S HOSPITAL (INCORPORATED): Resident Medical Officers (3).

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

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